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GAS BACILLUS INFECTION
WITH SPECIAL REFERENCE TO THE THERAPEUTIC VALUE OF THE X-RAY*

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Contrary to the opinion of many authors, gas bacillus infection is not infrequently seen in civil practice. At a state institution as Charity Hospital the surgeons observe many such cases, some possibly are unavoidable, but the majority the result of carelessness and improper management.

Our primary purpose in making this report is to stress the importance of the proper management of patients with wounds, which are potentially infected with anerobic organisms. Secondly, we wish to confirm the work of Kelly,[40, 41, 42] Faust[22, 23] and others, regarding the therapeutic value of the x-ray in gas bacillus infection.

HISTORY

Although Maisonneuve,[49] in 1853, is usually given credit for the first authentic description of gas bacillus infection, many authors prior to his time have alluded to the condition. Hippocratic writings[28] suggest the recognition of this "strange malady," as early as 300 B. C. Celsus is believed to have known of the occurrence of gas infection in pregnancy. In his monograph on gangrene (De gangreno et sphacelo, 1593) Fabry of Hilden[28] was the first to recommend amputation above the diseased part. De la Peyronnie is given credit for a fairly accurate description, while Quesnay in 1745 wrote on "Gangrene of Putrid Dissolution of the Humeral Mass." In the latter part of the eighteenth century, de la Motte and Kirkland mentioned cases. In 1822 Ollivier[28] produced experimentally hospital gangrene by auto-infection.

History reveals that during the various wars knowledge of the subject increased rapidly. Thus Larrey[28] during the Napoleonic war, Dupuytren[28] in the next period, Salleron and Pirogoff during the Crimean War made important contributions. It was, however, during the World War that the most valuable work was accomplished. The records of the medical corps of the various countries as well as personal reports make this period unequivocally the one during which the greatest advance in this disease occurred.

With Pasteur's discovery and isolation of the vibrion septique and the subsequent investigations of Koch, Novy, Welch and Nutall, the causative organisms were demonstrated.

ETIOLOGY

In gas bacillus infection the causative organisms, Cl. welchii group, and the predisposing factor (trauma) are absolutely interdependent. A large variety of organisms of the clostridium group has been isolated, but in the literature three species are mentioned most frequently:

1. Clostridium welchii (B. perfringens: B. aerogenes capsulatus) Welch and Nutall, 1892; 80 to 90 per cent of cases.
2. Clostridium oedematis maligni (vi-

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brion septique), Pasteur, 1881; 10 to 25 per cent of cases.

3. Clostridium oedematiens, Weinberg and Sequin; 5 to 15 per cent of cases.

Eliot and Easton[20] state that in this country any other organisms than Cl. welchii are rarely identified. In our experience these findings were similar. Vincent[67] believes that the "maximum activity of the microbes of gaseous gangrene results from their combined action." Grantham[35] contends they live in symbioses together as well as with the pyogens and Cl. tetani.

They are widely distributed in nature. Rural sections are reported to be more contaminated than urban. This is largely due to the dissemination of fecal material by cattle, that harbor these organisms in the alimentary tract. However, they are not only "soil" organisms but can be cultivated from our immediate environment. Gage[26] and Maes[48] emphasize the frequency with which gas bacilli can be cultivated from wool, with a special reference to clothing. Gage[26] has cultured the organisms from wadding of shotgun shell in every attempt.

The higher incidence of gas infection in the negro is reported by Veal[66], Eliason and Erb[19] and Boland[3]. The latter explains this occurrence by the poorer hygienic state of that race.

Destruction of muscle with liberation of carbohydrate in deep closed wounds forms ideal growth conditions for these anerobes. In most reports, compound fractures carry the greater incidence of gas bacillus infection. Next in order of frequency are deep penetrating wounds of muscle, especially those inflicted by shotgun. According to the Surgeon-General's office, only 1.08 per cent of injuries to soft parts ever developed the infection, while more than 5 per cent of the compound fractures were complicated.

The occurrence of this infection in "clean" postoperative cases has been a source of interest and worry to every surgeon. Warfield[70] directed attention to the frequency of this complication in amputations, especially in diabetics and suggests a prophylactic dose of antitoxin in such cases preoperatively.

Robert, Johnson and Brukner[19] were able to cultivate Cl. welchii from the skin of 21.7 per cent of unprepared abdomens; 8.3 per cent from knife blades used in making the incisions and 9.2 per cent from the peritoneum when opened for non-inflammatory conditions. The occurrence of such infections following appendectomy and cholecystectomy has been reported by Johnson[39], Orr[57], Ochsner[58], Gordon[32], Gamble[27], Howe[38], and others.

PATHOGENESIS

Gas bacilli injected into living and untraumatized muscle do not grow, but when trauma occurs, and when the wound is open some of the clostridium group are usually present. Of 216 wounds examined by Taylor,[64] 130 (61 per cent) showed the presence of gas producing organisms. Of the latter, only 45 (35 per cent) exhibited clinical symptoms. Bohlman's statistics are identical, only one third of the wounds contaminated ever developed gas bacillus infection.

Nevertheless, Cl. welchii and allied groups, when present in an ideal medium, waste little time in producing pathology. The affected muscle is at first dull and opaque, brick red in color and resembles cooked meat. It does not contract, nor does it bleed when incised. Soon it softens and becomes diffluent and changes in color to green, brown or black. The exudate is blood tinged. Gas is formed and forces itself up and down the muscle bundles, opening up new fields for infection. At first, the gas is within the muscle sheaths, but soon it ruptures into other fascial planes, and courses along the vessels and nerves. The production of the gas is explained by Thorek[65] as the result of fermentation by these organisms of dextrose and isomaltose which is converted from the glycogen of the living muscle. They are putrefactive organisms as well and the gas according to Vincent[67] is the result of their combined action. The most virulent organism alone may produce little gas. The odor is very offensive, supposedly quite characteristic,

and described as "mousy" or "decayed meat," and is probably due to the action of saprophytes.

This gas may develop a high pressure. In testing the force the gas may produce, Taylor's[64] experiment revealed that within two hours the pressure begins to rise and in six hours it may reach a pressure of over twenty-three pounds. Manson[54] states he has obtained a pressure of thirty pounds in twenty-four hours' incubation. Thus there is further destruction of muscle through stoppage of the capillary circulation of gas emboli and pressure. Later larger non-anastomotic arterioles are involved. Finally, the main arterial trunk may be occluded, especially if the wound is closed or enclosed in a rigid cast.

The group of anerobes, termed clostridium, produce powerful exotoxins, which vary according to the virulence of the organisms. The same organisms may vary in this respect under different circumstances.

Glenny, et al,[31] discovered multiple toxins produced by some of the organisms of the Cl. welchii group. Clinically, we recognized two or possibly three distinct toxins: One necrotic, causing local destruction; another hemolytic causing a severe anemia; and finally a pure systemic toxin. The latter two may easily be the result of a single toxin.

Vibrion septique and Cl. oedematiens produce the more powerful toxins, especially the former, which according to Ghormley[29] has been known to produce local edema with no gas and a fatal toxemia.

Therefore, the following clinico-pathologic classification is submitted dependent upon the relative degree of the various toxins present, the extent of local pathology, and the relation of the lesion to the severity of the systemic toxemia.

1. Primarily toxic (fulminating): The patient is extremely toxic and in shock, far out of proportion to the local lesion. Death will occur in a few hours if effective therapy is not instituted immediately.

2. Primarily gangrenous (mechanical): In a few cases the entire extremity may be gangrenous and yet the patient suffers a relatively mild toxemia. Usually the existing pathology is pressure occlusion of the main trunks (venous and arterial) with little absorption.

3. The usual or common type: Occurring in neglected wounds in 12 to 24 hours. In these cases the degree of toxicity is in proportion to the local lesion.

4. Localized or delayed: A slowly developing lesion with a mild systemic reaction. May occur even weeks later. Odor, gas or culture warrants the diagnosis.

DIAGNOSIS

The establishment of a diagnosis of this disease is relatively simple and the recognition of the early cases is not difficult, if the condition is kept in mind. All potentially infected wounds should be scrupulously examined if an early diagnosis is to be made.

Most authors agree that pain of severe intensity and out of porportion to the lesion is probably the earliest if not the most constant finding. The pulse is very rapid in relation to the temperature. The tachycardia is evidently dependent upon the toxemia, the bacterial toxin having a specific direct effect upon the heart muscle. The anemia, caused by a hemolytic toxin, is characteristically present according to Eliot and Easton[20]. In addition, there is also general malaise, marked prostration, and restlessness, as well as apprehension.

The presence of gas infection can be detected before the onset of marked systemic reaction except in the extremely toxic cases. Dolor, rubor and tumor are usually the first signs complicating the wound. Swelling, with exquisite tenderness and diffuse redness about the wound, is characteristically early. Slight pressure about the edge of the wound will express a sanguineous or watery discharge, with or without a few bubbles of gas. Direct smears will reveal the classical gram positive, non-motile, encapsulated bacilli in considerable numbers together with a few staphylococci and streptococci. This is absolutely diagnostic. To wait for positive cultural findings is useless and far too dangerous.

Ghormley[29] is of the same opinion and states that smears are more valuable than cultures.

Biologic tests and "stormy fermentation" (Robinson and Stovall[60]) of milk, give rapid information, but it should be recalled that the proof of the activity of the organisms in vivo by their activity in vitro occurs at the expense of the host.

In the past such diagnostic criteria may have seemed inconclusive for the institution of therapy, especially when amputation was resorted to immediately, but in this day of less radical methods such findings are absolutely adequate. If a diagnosis is not confirmed by culture, no harm has been done. The wound was rightly treated as an infected wound with the addition of other more specific remedies.

Palpatory and auscultatory crepitation and the easily recognized odor are relatively late findings. The fulminating case is most spectacular and these findings, although classical, are sometimes misleading. The patient is in a state of profound shock, with a rapid, thready pulse, a delirious state and the temperature varying from 103° to 105°. Prompt therapy must be instituted if the life of the patient is to be saved. Following traumatic injuries the diagnosis is not puzzling, but gas bacillus infection as a postoperative complication is frequently confusing. In the latter condition the analytical power of the surgeon is really tested. He is at a loss and believes that the symptoms are dependent upon occult bleeding. Gamble[27] emphasizes the erroneous diagnoses of the so-called "liver deaths" or hepatic insufficiency following cholecystectomy, that eventually prove to be gas bacillus infection of the wound.

The primary gangrenous type due to mechanical occlusion of the main vessels is late and is clinically obvious. An entire extremity is mortified and gaseous with relatively mild systemic toxemia.

The localized or delayed case is usually recognized by x-ray, incision and evacuation and by culture. There is little systemic reaction.

X-RAY DIAGNOSIS

Early in the World War the importance of the use of the x-ray in diagnosis was emphasized. The value of the x-ray as one of the earliest positive findings is stressed by Rhinehart[61] and Lingeman[46] who have seen positive gas findings in infected wounds as early as two and one half to four hours. As small an amount of gas as 1 c. c. has been demonstrated by the x-ray experimentally.

Gas shadows in the roentgenograms are recognized as round or oval areas of lessened density, or as dark streaks either under the skin or extending between the muscle bundles or under fascial planes. The x-ray technic for demonstration of gas should be of a high milliamperage and of relatively low voltage to give good contrast in the soft tissue. The usual bone detail technic often burns out the gas shadows.

The presence of gas shadows in an x-ray film alone without clinical evidence of infection is of no value in the diagnosis of this disease. Air may, and often does, enter wounds. In compound fractures air is frequently sucked into the wound on reduction of the fragments. Air may be driven into gunshot or penetrating wounds. Again in amputation, air is frequently present under the skin flaps soon after operation. Wounds penetrating or in proximity to a joint may show gas shadows, because of the sucking action on motion of the joint. Hydrogen peroxide used in cleansing penetrating wounds almost always produces gas shadows in the x-ray films taken immediately thereafter. These various accidental air shadows will tend to disappear in six to twelve hours on successive examinations. In contradistinction, the classical x-ray shadows of gas infection will spread and increase in amount.

MORTALITY

In the literature the larger groups of collected cases are interesting but give no information of the advanced improvement in the management of the disease. In Millar's[55] authentic report of 607 cases in civil life, (during the interval from the post-Lister period up to 1930) the mortality in

these cases of 49.7 per cent is compared to that of the American Expeditionary Forces in France of 48.5 per cent. Of 5,449 cases collected, Manson[54] found a death rate ranging from 15 to 60 per cent with an average of 38.5 per cent. The British Expeditionary Force reports a mortality of 20 to 50 per cent.

In an attempt to determine the improvement in mortality rate, three periods were compared: (1) pre-war; (2) post-war (antitoxin); (3) recent. Prior to the World War most of the reported mortalities are in the vicinity of 50 per cent. Welch reported 59 per cent in 1900, while Cramp reported 48 per cent in 1912. In 1914, Chalier gives a death rate of 67.4 per cent. However, some of these were war cases.

With a better knowledge of the disease, improved surgical methods, and the universal use of antitoxic serum during the next interval (post-war period) there is some reduction in the death rate. Boland[8] reported 67 cases with a mortality of 32.4 per cent, which was reduced to 15.4 per cent with the use of adequate dosages of serum. Stone and Holsinger[62] give almost identical findings. In 52 patients, 17 died, a mortality of 32.4 per cent; while in 29 patients treated with serum, a death rate of 15.3 per cent was reported. Ghormley[30] compares a 57.5 per cent with 13.4 per cent following the use of serum. Similarly, Bates[2] reduced the mortality from 56 per cent to 12 per cent. Veal[66] reported 27.7 per cent mortality for cases at Charity Hospital. He finds a great reduction in mortality with the use of serum.

In the "recent period" with the valuable work of Kelly et al[42] on the therapeutic use of the x-ray a still greater reduction in death rate was found. Not only was the mortality lowered, but many limbs saved which in the past would have been sacrificed. In his group of collected cases the mortality ranged from 5 to 10 per cent dependent upon the type. In our series, we treated twenty patients with two deaths, a mortality of 10 per cent.

A critic may think that those interested in this work are too enthusiastic over a mortality based on so few cases. But when one witnesses the spectacular effect of therapeutic x-ray in gas infection he will realize that its value is not over estimated and that the method is well worthy of further study.

TREATMENT

The primary aim should be to prevent the development of this disease. In no other phase in surgery is prophylaxis more important than in traumatherapy.

Wounds of any severity are invariably associated with some degree of shock, the treatment of which is paramount. Operative surgery of any type is absolutely contraindicated under such circumstances. The control of hemorrhage and the protection of the wound with sterile dressings is essential until the general condition permits thorough local treatment.

The immediate care of wounds is the chief factor in reducing the incidence of gas bacillus infection. Antitoxic sera and x-ray are valuable adjuncts in the prophylaxis, but they will never replace correct surgical measures.

With proper knowledge of the pathogenesis, the surgery should include such measures that will render the wound relatively free of organisms and devitalized muscle tissue upon which they feed.

PROPHYLAXIS

The proper management of wounds should be governed by the following rules:

1. Do not delay: Treat the wound as soon as possible. Late wounds are treated in a similar manner except for closure and after care.

2. Use general anesthesia: Avoid the use of ether because of the additional heart damage.

3. Preparation of field: (a) Carefully shave and wash the part, using green soap and water; (b) irrigate wound with large quantities of normal sterile saline; (c) antiseptics around, but not in the wound.

4. Complete debridement: (a) Strict operating room technic; (b) remove all foreign material, irrigate at intervals; (c) excise all devitalized muscle tissue; (d) careful hemostasis with silk No. 1.

5. Repair of wound: (a) Close tight if absolutely certain of no potential infection; (b) leave wound wide open and irrigate, if in doubt.

6. Fracture with open wounds (compound fractures): (a) Wound handled as above; (b) remove all small bone fragments without a blood supply, and clean contaminated edges of larger fragments with rongeur; (c) reduce or interlock fragment if possible with slight manipulation; (d) transfix fragment, using suitable splints rather than plaster for fixation in order to make frequent careful inspection of the wound; (e) leave wound wide open.

7. Administration of prophylactic antitoxin: Polyvalent gas antitoxin and tetanus antitoxin.

8. Preventive x-ray: Especially in older and suspicious wounds.

The rules are mostly self-explanatory, but slight emphasis on a few points is essential. The use of plaster casts and tight dressings should be avoided. In compound fracture the double pin fixation as described by Caldwell[6] should be used. Whether closed or opened wide the wound should be meticulously examined at frequent intervals.

The value of prophylactic antitoxin is undisputed when given in large quantity. Veal[66], Ghormley[29] and others showed its beneficial effect. The preventive use of x-ray is a recent development and has not been reported. We have treated eight cases, with no occurrence of the disease. But, of course, such a small series is of no significance.

Formol-toxoids in the prophylaxis of gas gangrene are advocated by Penfold and Tolhurst[59], who state that men in the army, women in child-bearing period, and laborers should be vaccinated.

The main pitfalls occur when one is not able to do such extensive procedures and "had to do the best under the circumstances." With the improvement of medical standards even in small communities we should hear that statement no longer in civil practice. Whenever one is not sure he has removed all potentialities, never close the wound, but open wider and irrigate with antiseptics.

Once gas infection has developed, we are dealing with a specific disease, the therapeutic indications of which are prompt and proper surgical measures. The treatment is based upon a working knowledge of the pathogenesis and symptomatology. We must "halt the pathogenesis and combat the symptomatology".

Many surgeons fail to realize that we are dealing with a patient as well as a wound. This individual is usually extremely toxic and in a state of shock of such severity that frequently radical methods are incompatible with life.

In most cases, "less radical" methods in conjunction with the aid of serum and x-ray are far superior. We prefer the term less radical to the word "conservatism," which should play no part in the treatment of this disease. There cannot be set rules; each case must be individualized. A keynote to success is that the surgery must be more extensive than the pathology. Decompression to prevent the progress of the destructive gas coupled with complete excision of the involved tissue is imperative. Muscle should be excised until there is active bleeding and contractility which indicate that the infected area has been passed. The wound is left wide open and irrigated with hydrogen peroxide. Intensive oxygenation of the wound has helped the local process as well as reduced the toxemia in the experiences of MacKechnie[47] and Drogoz[17].

The consensus of opinion in the literature regarding the use of therapeutic antitoxin is fairly uniform. It is a very valuable adjunct in the treatment of this condition. The larger the dosage, the better are the results obtained.

There are many monovalent and bivalent antitoxic sera. The one in use at Charity Hospital is bivalent for vibrion septique and Cl. welchii. Vincent[67] has justly emphasized the necessity of polyvalent sera, as the pathology is due to the combined action of both the saprophytic and pathogenic anerobes of the Cl. welchii group. No

doubt some of the poor results obtained are due to inefficient antitoxin.

The use of sulfanilamide in these cases has been reported with success by Bohlman[4] and others. Spray [63] experimenting in vitro with the drug, questions its efficiency against Cl. welchii. Dawson[13] obtained excellent results with auto filtrates. We have had no experience with either remedy.

X-RAY THERAPY

The beneficial effect of the x-ray in inflammatory conditions[15] is generally accepted to be of value and is frequently employed in the treatment of furuncles,[10] carbuncles,[10] erysipelas,[15] pneumonia,[56, 18] trachoma,[50] parotitis,[37] and early mastoiditis.[33, 34] The use of x-ray therapy in the treatment of gas bacillus infections, however, is relatively recent and has been tried in too few cases, and by too few surgeons for the method to be of definitely established value. It is our hope that the addition of these cases to those already reported will furnish more evidence in its favor and stimulate others to try x-rays as an aid in the prevention and treatment of gas gangrene.

In 1928 Kelly[40] introduced x-ray as an aid in the treatment of gas gangrene. His preliminary report of six successfully treated cases was in 1933. Faust[22, 23] and Hanchett[36] followed with successful results.

In 1937, Kelly and Dowell[42] presented their results of x-ray therapy in 138 cases of gas gangrene, collected from physicians in this country in answer to a specific questionnaire. They reported a total mortality of 10.8 per cent including arteriosclerotic and diabetic cases which carried a death rate of 55 per cent. In typical gas gangrene, the mortality was 7.7 per cent. Their results also showed that the mortality in amputated extremity cases was twice that of the non-amputated extremity group. In this series, 18 patients received no serum, and all but one recovered.

Since it has been shown that the x-ray has relatively little or no effect on Cl. welchii in vitro, the question is always aroused as to the explanation of the beneficial results obtained in vivo by x-ray therapy. Although no definite scientific proof has been offered as yet, the clinical result speaks for itself. There are, however, two or possibly three plausible theories. Faust[22] suggests after the experimental study of the effect of x-rays on tissue (Stanford University) "that the x-rays on nutrient fluids is deadly to protozoa by producing small quantities of hydrogen peroxide."

Another theory is the liberation of a proteolytic factor which may be resistant to the toxin of the gas bacillus, and a third, is the increase of local resistance due to the sudden liberation of enzymes from leukocytes killed in mass by x-irradiation.

TECHNIC

The x-ray factors used at Charity Hospital in treating gas infection are as follows:

Voltage	Extremity cases, 90-120 kv. Trunk cases, 200 kv.
Milliamperes	Extremity cases, 5 ma. Trunk cases, 20 ma.
Filtration	Extremity cases, 3 mm. aluminum Trunk cases, 0.5 mm. copper 1 aluminum
Skin Target Distance	30-50 cm.
Frequency of Treatments	Early cases, every 12 hours. Late cases, every 8 hours
Dosage	From 50-100 r. per area per treatment

The preventive technic in potential gas bacillus infections is similar to that used for early cases. While all the cases in this series were treated on stationary therapy units, similar results may be obtained on extremity cases with portable x-ray units. This is of distinct advantage in treating patients too ill to be moved.

REPORT OF TWENTY CASES

Nature of Injury		No. of Cases	Percentage
Compound Fractures		8	40
Auto-train	5		
Gunshot	3		
Penetrating Wounds		6	30
Gunshot	3		
Stab wounds	2		
Nail puncture (diabetic)	1		
Postoperative		5	
Amputation (Traumatic, 2) (Arteriosclerotic gangrene, 1)	3		
Osteotomy	1		
Abdominal	1		
Snake bite (incision and drainage)		1	5
Surgical Treatment			
Procedure:			
Less radical decompression and debridement	6	6	30
Decompression alone	7	7	35

Radical amputation			
Immediate	3	3	15
Delayed	3	3	15

N. B. One patient received no surgical treatment because of moribund state. Died six hours later.

MORTALITY IN RELATION TO INJURY

Type of Injury	No. of Cases	No. of Deaths	Mortality Rate Per Cent
Compound fractures	8	1	12
Penetrating wounds	6	0	0
Postoperative	5	1	25
Snake bite	1	0	0

In considering the mortality with reference to the type of injuries we find that our results are in agreement with other reports. The greatest mortality is in postoperative cases, 20 per cent. The one death in this group is not a fair test of the efficiency of the therapy. The patient was 65 years of age and his leg had been amputated for arteriosclerotic gangrene. He developed the infection postoperatively, but we did not see the patient until seven days after amputation. At this time, he was moribund and died six hours later. There was one x-ray treatment given.

In compound fractures our mortality is slightly higher than others reported. The fatality of this group, however, was a neglected case, admitted to the hospital seven days after his accident. The wound had been tightly sutured and was still closed when the patient was admitted. He was delirious, extremely toxic and in profound shock. Decompression alone was done. He received a large therapeutic dose of antitoxin and three x-ray treatments, but died thirty hours after admission.

SUMMARY AND CONCLUSIONS

We have reviewed the recent literature on gas bacillus infection and emphasized some of the more important phases of the disease.

Contrary to the opinion of many authors, gas bacillus infection is not infrequently seen in civil practice.

In this country, Cl. welchii is the most common offending organism. Compound fractures are complicated with gas infection five times as frequently as injuries to soft parts, according to the literature.

An accurate knowledge of the pathogenesis and symptomatology in this disease is absolutely essential for the early diagnosis and treatment.

An early diagnosis can be made in all cases if we only suspect and examine for the presence of the infection. Pain is an early constant finding. Smears are diagnostic and more valuable than cultures, because of the time involved in the latter. X-ray gives early positive findings.

A study of the mortality in the disease reveals that since the World War, with better surgical methods, the universal use of antitoxin, and recently, the therapeutic value of the x-ray, there has been a gradual decrease in the death rate.

Proper prophylactic management of all wounds, especially compound fractures, will lower the incidence of gas bacillus infection. Antitoxin and x-ray are both valuable preventive measures.

The best results are obtained with "less radical" surgical methods, adequate dosage of antitoxin and the therapeutic x-ray.

Twenty cases of gas bacillus infection are analyzed with a mortality of 10 per cent. In compound fractures, the death rate was 12 per cent, while in postoperative cases it was 20 per cent. This is in agreement with other reports.

We wish to emphasize that gas bacillus infection can be prevented, and with the recent methods offered, not only will many lives be saved, but many limbs as well.

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DISCUSSION

Dr. Mims Gage (New Orleans): I have been interested for some time in the subject presented by Drs. Charbonnet and Cooper. I sincerely hope that their observations as well as others will bear fruit and that we will have an additional safeguard in the treatment and cure of gas bacillus infections by the judicious use of the x-ray.

The Bacillus welchii is widely distributed in nature being found in the excreta of practically all animals (specially the herbivora), in the soil, and in all woolen goods. I have investigated woolen materials, such as gunshot wads and wool clothing, bacteriologically and have found the presence of B. welchii in 100 per cent. Experimentally, I have produced B. welchii in dogs and rabbits by traumatizing the muscles of the thigh and then introducing pieces of gunshot wadding and bits of wool clothing into the wound. This is of use clinically because all wounds of a traumatic nature involving muscles, which contain either gunshot wadding or bits of wool clothing, will certainly develop gas bacillus infection.

The Bacillus welchii is a sugar feeder; therefore it is necessary to have tissues traumatized in which there is a large amount of sugar present. We find such tissues containing a high content of sugar in the muscle, liver, blood clot, and to a less degree in the brain. As the organism is almost entirely an anerobe, it is necessary that a traumatized wound contains either devitalized muscle or blood cloth or both, and must be of an anerobic character. When this combination is present in traumatized wounds, the incidence of gas bacillus infection will be rather high. The wounds therefore that contribute to the development of gas bacillus infection are traumatized wounds with lacerated and devitalized muscles which occur in all compound fractures, especially those of the long bone. From these observations one can readily state that all lacerated wounds in which there is hematoma formation or devitalization of muscle are potential cases of gas bacillus infection. There is no type of wound that creates these favorable conditions more than compound fractures. Several years ago, Dr. Muir Bradburn cultured all cases of compound fractures that came into his service and found gas bacillus contamination present in 50 per cent of them.

Knowledge of these factors should convince the surgeon that he has in his power the methods that will prevent the development of gas bacillus infection. This prevention or prophylaxis consists of thorough debridement of all lacerated, contused wounds, gunshot wounds, and compound fractures. If the wound can be thoroughly cleaned mechanically, then primary suture can be done. If the cleaning is not thorough, it is advisable to leave the wound open widely in order that there will be no anerobic areas. An anerobiosis of the wound can also be overcome by the use of chemicals that liberate large quantities of oxygen. We have found that the instillation of peroxide of hydrogen into the wound every two hours is a very useful method in producing oxygenation of the wound. To wait until there is gas formation in the tissues, which can be demonstrated clinically or roentgenographically, is one of the greatest of surgical errors.

If the patient enters the hospital with well-developed gas bacillus infection, the surgeon is confronted with a very serious problem. In this type of case, the treatment consists of adequate exposure and removal of all devitalized tissue in the area of infection. The wound should be left open to produce aerobiosis, and chemical oxygenation should also be part of the treatment. Large doses of polyvalent antigangrenous serum should be given and repeated every twelve to twenty-four hours. Because of the rather high mortality in this type of case, x-ray has been used by a large number of surgeons and radiologists with most encouraging results. It is not easy to explain the beneficial result following x-ray treatment in this type of case. It probably is dependent upon the conversion of the vegetative forms of the gas bacillus into the inactive spores. This change of environment is probably dependent upon chemical changes that take place in the tissue and the exudate as well as the organism produced by the x-ray.

The authors have presented a series of cases tonight in which x-ray seems to have been of great benefit as the infection has subsided and the patient recovered. In some of the cases shown tonight, gas was demonstrated in the thigh after amputation of the leg and this was considered to be gas bacillus infection. It is a well-known fact that gas generated by the gas bacillus organism at times reaches the astounding pressure of twenty pounds to the square inch in the tissue. It is therefore readily conceivable how the gas may extend in the tissue a considerable distance from the infection. There have been cases of gas baccillus infection of the muscles of the calf of the leg which was limited to this area. Nevertheless, gas was demonstrated roentgenographically in the tissues of the thigh as well as the abdominal wall, although no B. welchii were present either in the thigh or the abdominal wall. Therefore, one must be very careful in drawing conclusions as to the presence of gas bacillus infection in the tissue made entirely on the presence of gas at these sites.

I would suggest to the authors that they use the x-ray prophylactic measure in the treatment of all severely traumatized injuries where there has been extensive muscle injury with and without blood clot formation.

Dr. Howard R. Mahorner (New Orleans): This presentation is very convincing and I think that it adds confirmatory evidence helpful in the management of these cases.

One thing I would like to call attention to is that it is so important to diagnose gas bacillus infection early and I quite agree that we cannot depend on the x-ray for diagnosis. The important thing about making that diagnosis is to be watching for the appearance of the infection. It can be detected by the fingers as early as by any other method. When there is potential gas bacil-

lus infection developing, the intern ought to examine the patient with sterile gloves around the wound and distal to the wound every six to eight hours. Whenever gas is present in the x-ray it is detectable by palpation.

There is another thing that I would like to call attention to and that is that remarkable results have been reported by the use of sulfanilamide in gas bacillus infection. Not that x-ray should not be used, for this presentation convinces one that it helps, but it is a very serious infection and everything we have to combat it should be used against it.

Dr. G. F, Fasting (New Orleans): I have been called upon to do quite a bit of the bacteriologic work reported in this paper and am responsible for the report of the presence or absence of Cl. welchii. There have been times when I would report absence and the doctor was convinced that there must have been gas. Other times, of course, we have found it.

It has just been stated, that you cannot rely upon the x-ray alone for diagnosis, but should include palpation. We have had two cases not so long ago, in which the laboratory reported the presence of Cl. welchii in routine pus examination. One was in a case of osteomyelitis with the wound open. The presence of Cl. welchii was not suspected. The wound being open and possible presence of granulation tissue may have prevented spread of infection and saved the patient. The point remains that gas gangrene bacilli were present, for which the laboratory takes credit in finding.

Another question to be considered is conditions simulating gas gangrene. I believe this occurs because in our attempt to isolate Cl. welchii, we have failed to show presence of anerobes. From the mixture of organisms present we were not able to conclude whether the amount of gas produced by them was extensive enough to penetrate high in the tissues. We are, however, sure of one thing, when a wound or leg has been amputated and small amounts of gas manifest themselves at the surface and we are requested to make a culture for gas gangrene bacilli, the laboratory representative will confirm the presence of bubbles but fail to isolate an anerobe.

This particular phase of bacteriology has not been adequately worked out. It is the wisest thing to be on the cautious side and assume gas gangrene bacilli present until proved absent.

Dr. L. S. Charbonnet, Jr. (In conclusion): We are very grateful to Dr. Gage and others who discussed our paper. Dr. Gage's suggestion, that the effect of the x-ray may be on the spores is logical and explains the beneficial prophylactic effect. We agree with Dr. Fasting that bacteriologic methods in regard to these anerobes are still in progress and not satisfactory as yet.

Finally, it must be emphasized that this infection can be prevented.

TUMORS OF THE ADRENAL GLAND IN EARLY CHILDHOOD

WITH A REPORT OF TWO CASES*

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INTRODUCTION

It has been my good fortune, in seventeen years of practice as a physician, to have had as patients two very interesting and rare cases of tumors of the adrenal gland with involvement of the kidney. The children, males, three years of age and twenty months of age, respectively, came to my office and were examined, operated upon, and left the hospital, all within a period of three weeks. One year has elapsed since operation, and, in each instance, these children are, apparently, in good health today.

The adrenal, or suprarenal gland—a flattened body situated in the retroperitoneal tissue at the upper end of each kidney, consisting of an internal medulla and an external cortex enclosed in a connective tissue sheath or capsule—was first described by Eustachius in 1564 and the adrenal medulla by Bartholin in 1666.

Tumors of the adrenal gland, clinically and pathologically, are an unusual finding. For clinical purposes, Tileston and Wolbach[22] divide these tumors, in children, into four classes: (1) those with metastases to the skull (Hutchinson's type); (2) those of simultaneous sarcoma of the liver and adrenal; (3) the type associated with precocious maturity; and (4) the tumors not falling under any of the preceding headings.

Player and Lisser[17] classify tumors of the adrenal into two main groups: (1) those of the medulla, and (2) those of the cells of the cortex. I shall discuss the subject under these two headings.

TUMORS OF THE MEDULLA

Tumors of the medullary portion of the adrenal gland are an uncommon discovery. The recorded cases are generally in children under six years of age and, in many instances, in children under three years of age. Among 22,000 admissions to the sur-

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gical wards of the Children's Hospital in Boston over a period of twenty-one years, Mixter[15] reports ten cases of neuroblastoma of the adrenal.

Until 1910 these growths were most frequently referred to as round cell sarcomas, lymphosarcomas, or gliomas. Wright[24] established their pathologic nature, and Pepper[16] and Hutchinson[10] described the clinical picture.

The "Pepper type" is characterized by early metastases in the liver (abdominal enlargement being an outstanding feature), and the early age at which the tumor appears. It may occur congenitally and is generally discovered before the third year. Pepper's series of cases were reported as "congenital sarcomas," with the site of the primary tumor in the right adrenal. Tumors located on right and left sides, respectively, have been recorded by Scott, Oliver and Oliver,[19] in a review of the literature. Kato and Wachter,[11] in a later summary to 1937, list twenty-six cases in the right and seventeen in the left adrenal. The classification frequently found today is "the Pepper type of neuroblastoma or sympathicoblastoma." McNamara[13] reports the tendency of this type to localization in the abdomen, but invading the blood vessels and lymphatics, although not forming distant metastases.

Hutchinson's tumor, so-called, frequently arises from the left adrenal medulla, but may be found in either suprarenal gland. Scott, Oliver and Oliver[19] give 8.5 years as the average age when tumors are discovered when primary in the left, and 3.5 years when primary in the right suprarenal gland. Appearance of metastases in the bones of the skull, and especially those about the orbit, is characteristic of this type, McNamara[13] describing the clinical picture as similar to that of an injury to the eye.

The attempt to distinguish between the Pepper and Hutchinson syndromes on the basis of location of the primary growth has, according to Askin and Geschickter,[1] proved untenable. They refer to the statistics compiled by Scott, et al[19] as showing that either side may be the site of the primary tumor in both syndromes, the most significant difference being one of age, . . . "average 12.7 months for the Pepper type compared with 6.9 years for the Hutchinson's syndrome. . . ". Kato and Wachter,[11] reclassifying the cases recorded by Scott, et al, and adding fifty-three cases reported between 1932 and 1937, list the average age of patient as follows: Pepper type, 1.7 years; Hutchinson type, 3.9 years; mixed type, 5.6 years; uncertain, 3.1 years.

Doub[5] says that late studies have shown the overlapping of the two types, and that they are indistinguishable pathologically and roentgenologically. Askin and Geschickter[1] observed the overlapping of the two syndromes in their series of cases. They state: "In cases in which the bones are involved, there may also be abdominal enlargement due to metastases to the liver, and enlargement of the regional lymph nodes may occur in either type. While anemia is a more pronounced feature with the Hutchinson's syndrome, severe secondary anemia may also occur in the advanced stages of the Pepper type." Kato and Wachter[11] believe that actually either type may produce both the skull and liver metastases in varying proportions.

Dunn,[5] and Lehman[12] have painstakingly reviewed the literature, analyzing case reports. Scott, Oliver and Oliver,[19] in their survey, bring the total number of reported cases to one hundred and sixty two. Kato and Wachter,[11] in reporting a case of adrenal sympathicoblastoma, with special reference to the biopsy of sternal marrow and of metastatic nodule in the skull, have studied the literature and are of the belief that some of the cases reported by Scott, et al, were ganglioneuroma rather than neuroblastoma and a few were lacking in data for positive identification. They add fifty-three cases of sympathicoblastoma in children, published since 1932, and their table No. 1 gives a summary of these cases. In a second table they summarize one hundred and seventy nine cases of adrenal neuroblastoma in children, reported from 1880 to 1937.

Neuroblastoma, sympathicoblastoma, sympathoblastoma, sympathogonioma, neu-

rocytoma are all terms used at this time. Meyer[14] believes that classification and identification of medullary adrenal tumors has been confusing as a result of the undifferentiated grouping of the cells.

From a physical diagnostic standpoint, Askin and Geschickter[1] believe it is important to observe that the retroperitoneal mass in the region of the kidney may be very large without producing renal symptoms and that there may be hepatic enlargement to a marked degree without jaundice or ascites, although there are exceptions to this rule. Their tables II and III, giving symptoms and findings in seventeen cases of neuroblastoma, are interesting. Microscopically, they believe that the recognition of neuroblastoma is difficult at times, it being impossible definitely to rule out, by the examination of a single microscopic section, lymphosarcoma and embryoma of the kidney of the Wilms' type.

In two cases of sympathetic neuroblastoma in children, Doub[5] reports that pain, referred to the spine and legs, was the initial symptom.

In the differential diagnosis, Mixter[15] states that neuroblastoma of the adrenals occurs with less than one-fourth the frequency of embryoma and that "occasionally a case is encountered in which there is rapid local growth of the adrenal neoplasm with no gross evidence of metastasis and in which the silent clinical course and the position of the mass simulate the true kidney tumor." Kato and Wachter[11] discuss the hematology of adrenal neuroblastoma and its differential diagnosis from chloroleukemia, acute leukemia, and lymphosarcoma. In a discussion following Doub's[5] paper, Sosman[20] speaks on differential diagnosis from a roentgenologic standpoint.

McNamara, Henneger, and Lytle,[13] and others, report the prognosis as grave, as most cases are diagnosed in advanced stages. However, the occasional case that has been cured by early removal makes the condition not entirely hopeless. These authors belive x-ray therapy of no apparent value, an opinion concurred in by Askin and Geschickter,[1] and Doub.[5] Doub used radiotherapy in two cases in children and believes it to be of no avail if there is evidence of metastases.

In preoperative procedure, Cecil[3] stresses the importance of determining the existence of a functioning opposite suprarenal gland before resorting to surgery on the offending side.

SURVEY OF CASES IN RECENT LITERATURE

Among the cases of neuroblastoma in children reported in recent literature are the following: McNamara, Henneger and Lytle[13] report the case of a girl, three years and seven months of age, first seen four months before death because of swelling of the abdomen. There had been no subjective symptoms and no fever. *Clinical diagnosis*: sarcoma of liver. *Anatomic diagnosis*: "Neurocytoma of the right adrenal gland; invasion of the right kidney and the inferior vena cava." This girl was the niece of another patient who died from carcinoma of the ovary.

Meyer[14] writes of a boy, three years old, who on admission to out-patient department had pain with swelling on the left side of the face, with some fever. One month later he returned and a diagnosis of neurocytoma of adrenal gland with metastasis was made; patient died one month subsequently. *Autopsy*: Abdomen revealed large tumor in right renal area. The anatomic diagnosis was right adrenal cell tumor with metastases to the skull, vertebrae, long bones, mandible, brain, ribs, and lymph nodes, which microscopically and sectionally, from one of the metastatic growths, was termed a neuroblastoma.

Askin and Geschickter[1] record seventeen cases of neuroblastoma of the adrenal occurring in children, their oldest patient being 13 years. Thirteen of the number were infants and children to six years of age. Males predominated with a ratio of 11:5; sex in one case not given. Tumors were equally distributed between the right and left adrenal; two were bilateral. All of the cases were fatal.

Rhea and Mead[18] report a fatal case in a five year old female, of adrenal neuroblastoma of the Hutchinson type, with primary bilateral adrenal tumor.

Their second case was a white female, three years old, who died three months after exploratory laparotomy and eight months after onset of symptoms. The diagnosis made by biopsy was that of a right sided tumor in the region of the adrenal gland with findings pointing toward a Pepper type of neuroblastoma.

Doub[5] relates two cases of sympathetic neuroblastoma in children, the initial symptom being pain referred to the spine and legs. The first case, that of a boy two and a half years old, gave a history of a fall from a tricycle four weeks previous to admission. The patient died five months later. The case was diagnosed by postmortem examination as sympathetic neuroblastoma of the left adrenal with metastases to lymph nodes, liver, lungs, and lumbar vertebrae.

His second case, a girl of four and a half years, gave a history of "pain in epigastrium and over the upper part of the thighs." Three months previously roentgen ray therapy had been administered by another physician for abdominal mass. Doub used roentgen therapy but child died two months later. Postmortem diagnosis was "sympathetic neuroblastoma arising in the medulla of the adrenals, with metastases to the pancreas, adjacent lymph nodes, liver and bone marrow."

Kato and Wachter[11] record a case of adrenal sympathicoblastoma, a male two and a half years old, who gave a history of a bluish discoloration about the eyes which first appeared three weeks before date of visit to clinic, first about the left and later about the right eye. There was no known history of trauma. Child died almost two months later. Case is classified as adrenal sympathicoblastoma (neuroblastoma), Hutchinson type.

They add in a footnote a second and later case of adrenal sympathicoblastoma (Hutchinson type) in a white female two years and three months of age, who died two and a half months after admission, in spite of one transfusion and eight x-ray treatments. Original complaint was swelling of left jaw, which first appeared three weeks previous to admission.

TUMORS OF THE ADRENAL CORTEX

Tumors of the adrenal cortex have been classified by Ewing[7] into: (1) hyperplasia, nodular or diffuse; (2) adenoma, and (3) carcinoma, according to Strohl.[21]

Hyperplasia, nodular or diffuse: " . . . the nodular type is usually well encapsulated, and frequently many areas of hyperplasia can be found in one gland. The diffuse type has recently been more commonly associated with the picture of hirsutism (Walters, Wilder and Kepler)[23] . . . Nodular hyperplasia may be associated with nodules in the liver, and sexual overdevelopment may be seen in this type of condition."

Adenoma: Ewing[7] has stated that true adenoma can be differentiated from hyperplasia by its atypical and neoplastic structure. Adenoma of the adrenal gland generally occurs singly, although there may be multiple and bilateral growths. Strohl[21] says that "the tumor frequently projects from the capsule as a reddish or yellow structure, which is well circumscribed. Hemorrhage and central softening may occur. Some adenomas weigh as much as four ounces and still retain the characteristics of a benign adenoma. On occasions the adenoma is invaded by fibrous tissue, to the extent that "it becomes quite firm and, as a result, is called a 'fibro-adenoma'. There is a tendency toward hyperchromatism of the nuclei in the true adenoma."

Carcinoma: This is the type of tumor that often produces secondary sexual changes.

Adrenal cortical tumors with endocrine dysfunctions are rare. The endocrine changes caused by tumors of the adrenal cortex vary greatly according to the age and the sex of the patient. Player[17] states that in girls, if adrenal cortical tumor is congenital or present in early infancy, it causes pseudohermaphroditism and in young girls such tumors cause, as Lisser terms it, "pseudo-precocious puberty." Successfully operated cases, with disappearance of the syndrome, have been reported by Collett,[4] Fordyce and Evans,[8] and Harris and Plieves.[9] According to Player, "In true

precocious puberty the clinical picture is due to ovarian hypersecretion in early life."

A case of virilism and hypertension associated with right adrenal tumor (carcinoma) of the cortical type, in a female two years old, is reported by van der Bogert.[2] The child died. Tumor mass was examined after operation; there was no postmortem.

Of true precocious puberty in boys due to adrenal cortical tumor with endocrine changes there are, up to the end of 1932, eight authentic instances recorded and confirmed either at operation or autopsy, according to Player and Lisser.[17] They add to this group their own case, a boy four years and eleven months of age, with astounding sexual precocity, from whom a left adrenal cortical tumor was successfully removed. The patient was alive and well twenty months after this operation.

As many fatalities occur during the first few hours after operation, Cecil[3] has suggested an operative technic designed to prevent shock.

The histories of my two cases of tumors of the adrenal are as follows:

CASE REPORTS

Case 1. On March 22, 1937, a boy, three years of age, was seen who complained of pain in the region of the liver. Upon questioning the mother, I found that he had previously been treated for enlargement of the liver with no results, and that for the past three weeks he was getting worse.

Examination revealed an undernourished, anemic-looking male child, weighing 24 pounds. Head, neck, and lungs were clinically normal. In the upper quadrant of the abdomen there was a hard, more or less regular, semi-painless mass which seemed to arise from the right kidney fossa, extending forward underneath the liver, down to about the level of the umbilicus. This mass, however, could not be differentiated and more or less separated from the liver and the lower margin seemed rounded. There were no abnormal findings, other than this. Blood pressure was 90/60. No cystoscopic or x-ray examinations were made; urine was negative in every respect. Blood examination did not reveal anything abnormal.

Believing that I had somehting unusual, I immediately called my consulting surgeon, Dr. E. S. Peterman. We conferred, and decided on a tentative diagnosis of tumor of the adrenal, and advised that it should be removed.

He was prepared for operation on March 23.

Operation: Abdominal incision was made over the tumor mass. Abdomen opened and a retroperitoneal tumor mass involving the entire right kidney, about the size of an ordinary grapefruit, was found. This was removed.

On March 29 the little fellow was sent home from the hospital and had an uneventful recovery.

Gross Examination: The globular tumor is approximately 3½ inches in diameter, and weighs 22 ounces. It is located within the capsule of the kidney, with only a small portion of the kidney remaining free. The portion of the kidney involved in the tumor is intimately incorporated, being gradually lost in the mass.

The incised surface is reddish yellow, interspersed with gray, translucent areas. A few hemorrhagic and necrotic areas are found in the portion more intimately connected with the kidney. The general appearance of the cut surface is variegated.

Microscopic Examination: The sections are composed of smooth muscle in which scattered mitoses are visible. This is supported by a delicate reticulum with a rather sparse vascular supply. No remains of the right kidney structure could be found, nor is there any visible attempt at glandular cellular arrangement.

The sections give the appearance of an embryonal myosarcoma.

Follow-up: Upon a recent check-up I found the boy weighing 40 pounds, he looks fine and there is no evidence at all of any abnormal conditions.

Case 2: On March 29, 1937, a male, 20 months old, was brought to my office. He complained of itching of the navel. Upon observation, I found a fairly well nourished, "pot-bellied" male child, weighing 29 pounds. Clinically, he was normal except for the large stomach, which his mother said he had always had, and that he had been examined on numerous occasions and given something for enlarged liver.

The red bug, the cause of the itching, was removed from the navel. On examination of the abdomen, I found a large tumor mass in the upper quadrant, extending from the kidney fossa to and forward under the liver, down to the umbilicus. Hardly believing that I could have another tumor of the adrenal and kidney, I had various examinations and tests made. The urine was reported negative. The blood picture, by Dr. S. R. Henry, was as follows: Erythrocytes, 6,250,000; leukocytes, 5,250; hemoglobin and coeff. 14.5 gm.—13—77 per cent; volume of packed red cells, 42 c.c.; color index, 0.8; mean corpuscular volume, 67 cu. microns; mean corpuscular hemoglobin, 31 per cent. The neutrophiles were: myelocytes, none; juveniles, none; stab forms, 3 per cent; segmented, 26 per cent; a total of 29 per cent. Shift to left (regenerative shift) none. Lymphocytes, 67 per cent; large mononuclears, 1 per cent; eosinophils, 2 per cent; basophils, 1 per cent; granulocytes, 29 per cent; total leukocytes, 100 per

cent. Malaria, negative. Hypochromia, negative. Poikilocytosis, negative.

No abnormal alteration in blood picture. Kahn test, negative.

After completing the above examination, I called a surgeon and we decided to operate.

Operation: The same surgical procedure as described in the first case was carried out and the little fellow made an uneventful recovery.

Gross Examination: The specimen includes the whole kidney and a tumor mass roughly oblong and nodular, measuring 4½ x 3 inches, and weighing 16 ounces. The color varies from the dark brown of the kidney tissue to a yellowish translucent appearance of the nodular masses. Hemorrhagic areas of various sizes are numerous, with the presence of cysts of different sizes.

The flattened kidney substance can be dissected over an area of about three-fourths of its surface, the balance being gradually incorporated and lost into the tumor.

The cut surface is very friable and necrotic in some areas, and containing areas of hemorrhage.

Microscopic Examination: The microscopic picture consists of adrenal cell (cortex) arranged in strands and in groups supported by a delicate stroma. The cytoplasm is granular and contains lipoid globules, giving the foamy appearance of an adrenal adenoma.

Follow-up: Since his recovery, I have seen him a few times, and I find the boy much finer and better in every respect. However, on a few occasions he has run fever and has had several stomach upsets which, I think, were probably due to pin worms, and lack of proper vitamins. He now weighs 36½ pounds and has had no signs of metastasis whatever. Due to the fact that the diagnosis was made, and he was operated upon early, he is well today.

SUMMARY

Two cases of adrenal tumor are reported occurring in small children.

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DISCUSSION

Dr. Charles J. Bloom (New Orleans): Dr. Morgan has presented a most interesting and rather unusual paper. He brought to your attention the fact that tumors of the adrenal glands are rather uncommon, and in a series of 22,000 children admitted to the Boston Children's Hospital, the incidence of renal tumors was 41, and of true adrenal tumors, 10. He also recapitulated the number of adrenal tumors which have been reported in the literature up to this present time, namely, there have been less than 215 cases reported in the literature. It is indeed most interesting that Dr. Morgan adds to this list of 215 cases, two additional cases of tumor of the adrenal gland.

Differentiation between the common types of tumors of the adrenals has been more or less classified into two groups: one, the Hutchinson type, and the other the Pepper. In the Hutchinson type, usually the left adrenal has been involved and when metastasis has occurred, it usually occurs to the skull. Whereas, in the Pepper type, usually the right adrenal has been involved, and when metastasis occurs, it usually involves the liver.

By presenting this case to this section, Dr. Morgan brings to our attention another possibility to consider when one examines the abdomen of a child and finds a tumefaction. To the findings of

the tumor must be added the more or less common symptoms associated with tumors of the adrenal, namely, elevation of temperature, vomiting, and abdominal pain, to which must be added, pallor, leukocytosis, reduction in the red cell count, and an enlarged liver.

Dr. H. O. Barker (Alexandria): We had a child who died recently in our hospital. The case was not diagnosed, but at autopsy it was found there was metastasis to the abdomen from a tumor of the suprarenal gland.

Dr. H. E. Menage (New Orleans): I am interested in the subject from a dermatologic standpoint and would like to know in what proportion of cases hyperpigmentation of the skin and of the mucous membranes was found.

Dr. Albert R. Morgan (In conclusion): Dr. Menage, I am unable to answer that question, because no pigmentation at all was found in these two cases.

The only thing I can suggest in closing this paper would be that routine examination sometimes reveals something unusual.

THE ELLIOTT TREATMENT OF PELVIC INFLAMMATORY DISEASES OF AMBULATORY WOMEN

ANALYSIS OF 73 CASES*

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The Elliott treatment regulator has been used in my office practice for the treatment of properly selected cases of pelvic inflammatory diseases of women for the past three and a half years with gratifying results. Seventy-three patients to date have been treated, and comprise this series upon which this report is based.

One of the foremost conditions to be fulfilled before the application of the Elliott treatment in pelvic inflammatory diseases of women in the office, is the proper selection of patients for treatment. It will be remembered that all cases must be ambulatory, and since many of the cases are acute, the acute stage must necessarily be treated in the home, in bed, along the following general principles:

1. Absolute rest in bed, both physical and physiologic; Fowler's position, as quickly as called to treat the case. Patient must not be allowed to sit up in bed, and all "wants" of patient fulfilled by a nurse or attendant.

2. Application of ice bag to lower abdomen during first two to four days followed by "heat tent" to pelvis, one hour on, at two to three hour intervals, during day, according to tolerance of patient. If patient complains of ice bag, one may use heat from beginning of treatment with gratifying results.

3. Vaginal irrigation may be given slowly once or twice daily, after the third or fourth day, using 1-5000 potassium permanganate solution at 120° for ten to fifteen minutes.

4. Instillation of 5 c.c. of 5 per cent mercurochrome solution in vagina after each irrigation.

5. The bowels should not be moved during first three or four days. The lower bowel may be emptied by daily enemata. Mineral oil may be given on third or fourth day. Diarrhea and constipation are to be avoided.[1] There is no better drug than paregoric or morphine for quieting peristalsis, and this should be used to quiet hyperperistalsis as this excites the already inflamed areas, many times spreading the disease.

6. Pain is relieved, in addition to the above, by use of sedatives, codeine or morphia.

7. Distention can be avoided and overcome by use of proper diet, heat tent, Levin tube,[1] rectal tube, and pitressin[1] or prostigmine.

8. No solid food should be given during first two to three days, but plenty of fluids by mouth and hypodermoclysis, or venoclysis, of ten per cent glucose in normal saline, in no less than 3,000 c.c. quantities in twenty-four hours. After first two or three days, high carbohydrate, low residue, light diet is followed.

9. Foreign protein, in the form of lactigen, is used in 10 c.c. quantities, given intramuscularly every two to three days. This does indeed favor rapid resolution of the exudate, especially when the cause is due to the gonococci.

10. Tonics are given when necessary.

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11. Operative procedures are generally forbidden unless localised accumulations of exudate are inevitable and rupture is feared. It is now known that with the Elliott procedure many cases of pelvic cellulitis and pelvic abscess will undergo rapid resolution and resorption.[1]

12. The case must have become quiescent, the temperature normal for four to five days, and patient able to be brought to office in ambulance or automobile before treatment can be begun. This usually takes about fourteen days from beginning of treatment.

13. Vaginal and rectal examinations are made at intervals of five to six days to note progress and development of complications.

Before one is able to make proper selection of patients for treatment, all of the general indications and contraindications for the use of the Elliott treatment regulator must be known.

THE INDICATIONS

1. Acute salpingitis (with or without tubo-ovarian abscess; with or without pelvic peritonitis.)
2. Subacute salpingitis.
3. Chronic salpingitis.
4. Acute pelvic cellulitis, with or without "frozen" or "fixed" pelvis.
5. Subacute pelvic cellulitis.
6. Postpartal and postabortal parametritis.
7. Pelvic abscess.
8. Pelvic thrombophlebitis[1].
9. Acute gonorrheal vaginitis and endocervicitis.
10. Cystitis.[1]
11. Proctitis.
12. Dysmenorrhea.
13. Erosion of cervix.
14. Senile vaginitis.

THE CONTRAINDICATIONS

1. Menstruation—not absolute; but better to delay treatment, especially if profuse.
2. Uterine hemorrhage—profuse.
   a. Postabortal.
   b. Postpartal.
   c. Tumors.
3. Severe secondary anemia, with or without emaciation. Not absolute, but rather to indicate caution should be observed in that routine Elliott treatments are weakening in these types of cases and may even produce "fainting spells."
4. Acute salpingitis—early. Not absolute, but mentioned inasmuch as, in many cases, the pain produced is intolerable during first few days.
5. Plastic operations—cervix, bladder and perineum, with much scar formation. Not absolute, but low temperatures must be used to avoid serious resorption of scar tissue.

INSTRUCTIONS TO PATIENTS

Having made proper selection of case to be treated, the following written instructions are given to the patient to be executed at home prior to coming to office:

1. Void, as completely as possible.
2. Take vaginal douche, very warm, 1-5000 potassium permanganate solution.
3. Take enema of soap water to empty lower bowel.

When patient arrives at office, she may be allowed to rest for one-half hour during which time the following is observed:

1. Be sure bladder and rectum are empty.[3]
2. Examine vagina to see if it is dilatable and swab to remove excessive secretions.
3. Take mucosal temperature of mouth, bladder, vagina and rectum before, and immediately after treatment.
4. Check Elliott machine to make sure it is in proper order, that the temperature of water is not over 115° F. to start. Time is saved by heating the water to 110° in sterilizer, or over bunsen flame, before pouring it into the tank of the treatment regulator.

To obtain the greatest possible benefit from the treatments, the following points should be noted:

1. The temperature of water, as registered, is gradually elevated at the rate of ½° to 1° per minute until tolerance of patient, or not more than 125° or 130° F.
2. The duration of each treatment depends to some degree upon tolerance of

patient. Usually the first treatment is 15 minutes; second, 15-25 minutes; third, 25-30 minutes; fourth, 30-45 minutes, and no treatment is ever longer than one hour.

3. The vaginal applicator should be distended to tolerance of patient, but never to cause discomfort. Usually one to three pounds of pressure is sufficient. This is not often sufficient completely to flatten the vaginal folds, but burns will not occur with guarded temperature. More pressure is needed in large, relaxed, roomy vaginas.

4. Frequency of treatment is every second to third day. This depends upon the severity of disease, and distance and manner of transportation to the office. In chronic cases, treatments can be given every day. Too much physical disturbance is inadvisable, as a flare up of the disease may result.

5. Check all temperatures of patient immediately before treatment and just before treatment is discontinued.

6. Inspect vagina "cautiously" after removal of applicator bag. If there should be a burn, treat topically, insert tampon of vaselin or boroglycerine[4] and allow to remain in four to six hours. Continue next treatment as usual, but at a lower temperature.

7. Allow patient to rest at least one hour after treatment before returning home.

8. Sexual intercourse is to be avoided for three months after discharge of patient.

9. Always request some one of the family, or relative, to accompany patient to office.

EFFECTS OF TREATMENT[5]

Vaginal examination immediately after treatment, reveals a very warm vagina with an increase in secretions. The entire mucosa is red or hyperemic. After two or four treatments there is softening, shortening, and broadening of cervix. After two to four treatments, symptomatic improvement is noted, due to a diminished degree of pain and tenderness in pelvic organs. After four to six treatments, the mobility of uterus is greatly increased and the adnexae more easily palpable. Some cases, of course, require longer periods of time to show improvement.

PHYSIOLOGIC EFFECTS OF INCREASED HEAT[6]

1. Has destructive action upon bacteria, especially gonococcus.

2. Increases rate and volume of circulation and causes vasodilatation.

3. Increases phagocytosis.

4. Increases local tissue metabolism.

5. Increases lymph formation.[7]

6. An increase of 17 per cent in leukocytes.[1]

7. Erythrocyte sedimentation rate: I did not use this test in any of these cases, relying on clinical findings to determine progress, but according to Dickey,[7] the rate is definitely reduced to normal as clinical improvement is noted. More value is placed on this than on the white blood cell count, Boland[1]; when the sedimentation time remained stationary, it was a fairly good indication that the case would not respond to the Elliott treatments. In these cases in which the sedimentation time dropped and the white count became elevated, good results were obtained.

When these physiologic changes are produced in inflammation of the pelvis, the result is[2]:

1. A prompt subsidence of congestion and inflammation.

2. Resorption of exudate.

3. A definite destructive action on bacteria, especially the gonococcus.

4. An out-pouring of mucus and washing away of infective organisms from cervix and vagina.

BURNS ARE DUE TO:

1. Too high and too prolonged temperature.

2. Rugae of vagina not being spread properly by incompletely distended bag so that local temperature is not uniform. This can be avoided in cases that cannot tolerate much pressure by lowering the temperature.

3. Skin of vulva and perineum is less resistant to heat than vaginal mucosa,

therefore must be protected or burns will result from neck of bag.

The seventy-three patients in this series were diagnosed and analyzed as follows:

TABLE 1

Diagnosis	No. of cases	Average age	Average duration of disease	Average no. of treatments	Temp. of water Min.	Temp. of water Max.	Pressure pounds Min.	Pressure pounds Max.	Duration of treatment in minutes Min.	Duration of treatment in minutes Max.	Results* Good	Results* Fair	Results* No. Imp.	Major Surgical
Group 1. Acute and subacute—specific	12	22	18 days	12	110	130	1	3	10	60	10	2	0	0
Group 2. Subacute—non-specific	14	23	32 days	15	110	130	1	3	10	60	10	4	0	0
Group 3. Chronic salpingitis	17	26	1½ years	22	110	130	1	4	10	60	7	10	0	8
Group 4. Postabortal parametritis	8	25	16 days	8	110	130	1	3	10	60	7	1		0
Group 5. Postpartal parametritis	8	22	28 days	6	110	130	1	3	10	60	6	2		0
Group 6. Endocervicitis	7	28	1 year (?)	7	110	130	1	4	15	60		4	3	4
Group 7. Vaginitis—senile	3	54	1 year	10	110	124	1	2	5	30		2	1	0
Group 8. Postoperative inflammation	4	25	3 mos.	6	110	128	1	3	10	30	3	1		0

*There were no injuries in this series.

GROUP 1. ACUTE AND SUBACUTE SALPINGITIS

In this group there were twelve patients in whom vaginal and cervical smears showed gram negative intracellular diplococci. These were treated at home, according to the plan outlined in the beginning of this essay, for the acute stage of pelvic inflammatory disease. I have always found the use of 5 c.c. 5 per cent mercurochrome solution instilled in the vagina to be very beneficial. Negative smears were obtained in these cases after the fourth week of treatment or after eight to ten Elliott treatments had been given. Topical treatments were continued, however, for one to two weeks longer. Two of these patients developed Bartholin abscesses in the second week which healed promptly after incision and drainage. Cul de sac abscess did not develop in any case. Since I used the combination treatments, mercurochrome instillations and Elliott treatments, I am unable to state the value of heat produced toward actual destruction of the gonococci. However the following temperatures averaged from five cases, from Group 1, in specific locations, with varying Elliott machine temperatures, are cited:

TABLE 3

TEMPERATURES REPORTED BY HOLDEN AND GURNEE[3] WHEN ELLIOTT TEMPERATURE IS 130°

Urethral meatus	104.0°
Bladder	104.2°
Anterior rectal wall	106.0°
Posterior rectal wall	104.2°
Cervix	112.0°
Vagina	103.0°
Body temperature	0.4° rise

Holden[1, 8] states that by means of the Elliott apparatus, a consistently uniform temperature of 130° can be maintained for any length of time against a large area of distended vagina, cervix, adjacent parametrium and pelvic organs.

TABLE 2

Location	Before treatment	Elliott temp. 115°	Elliott temp. 120°	Elliott temp. 125°	Elliott temp. 130°*
Oral	99	98.8	99	98.6	98.2
Urethra	100.2	103.8	103	102.3	102.5
Bladder	100	102.1	102.8	101.5	101
Vagina**	100				
With Bag		109.9	112	113	115
Without Bag		101	104	103	106.3
Rectum	100	102.8	104.6	104	103
Folded bag—distended		114	119	123.5	128

*Elliott temperature of 132° plus was not tolerated.
**Vaginal temperatures taken immediately after removal of bag.

Holden and Gurnee[8] report that gonococci are killed in a few hours by a temperature of 106° F. F. J. Van Putte,[9, 10] Stockholm, has cultivated the gonococci for indefinite periods at 106.7° and has found most strains viable after exposure for six hours to 113°. This tends to discredit Holden and Gurnee's[8] report. Holden and Gurnee also state that the gonococci are destroyed, in a few minutes, after exposure to 122°. This is undoubtedly true, but analysis of Table 2 shows that when the Elliott machine temperature is 130° F., only an average temperature of 115° F. could be maintained in the vagina. These temperatures were taken between the bag and vaginal mucous membrane. I know a temperature of 130° F., as reported by Holden and Burnee,[1, 8] cannot be maintained in the vagina.

This is further supported by Herring's report[10] which demonstrated that a temperature of 122° in the vagina cannot be maintained by use of the Elliott machine. He states that a temperature of 100° in cervix and 114-121° on vaginal surfaces is all that could be maintained in his experience with the Elliott machine, using a galvanometer connected with a thermocouple to secure findings. I agree with Herring's report. The maximum vaginal surface temperature obtained in this series (table 2), when the Elliott machine temperature was 130° F., was 116.2° F. Temperatures higher than 131°, as registered on the Elliott machine, cannot, generally, be tolerated.

It is also reported by Holden and Gurnee[8] that there is a 0.4° rise in body temperature during treatment with the Elliott machine. Table 2 shows definitely a decrease in oral temperature, at the end of a treatment, variable between 0° and 0.8°. Only occasionally will there be a rise in oral temperature.

The average bladder temperatures are generally approximately one degree lower than urethral temperatures due to the fact that it is a cavity containing a variable amount of urine.

The temperatures of the vagina, with the bag, vary in direct ratio, with the Elliott temperatures, averaging ten degrees less. One to two degrees is due to the cooling effect of the tubes and bag, while approximately six to eight degrees is due to the cooling effect of the tissues and the vascular systems.

The temperatures of the vagina, taken immediately after removing the bag at the end of a treatment, averaged approximately 9° F. less than with the bag. Rectal temperatures vary according to the amount of intestinal content collecting during treatment.

All of which lends support to the statement that it is doubtful if tolerable temperatures obtained with the Elliott machine are sufficient to be lethal to all strains of the gonococci.

It will be noted that the results of Group 1, Table 1, were good in 10 cases and fair in two cases, with an average of twelve Elliott treatments. This number is four to six less than generally reported for such cases, but I frankly believe this is accounted for in the plan of treatment for the "acute stage." Resolution is therefore more rapid and more complete.

GROUP 2. SUBACUTE (NON-SPECIFIC)

Represents 14 patients, eight of whom gave a suggestive history of Neisserian infection, and six of undetermined origin. The results were about the same as in Group 1.

GROUP 3. CHRONIC SALPINGITIS

Seventeen cases are presented in this group. The results are not so striking as shown in Groups 1 and 2, but the reason is undoubtedly due to the fact that in Groups 1 and 2, the "disease" had to be dealt with, while in this (group 3) the "results" of disease were dealt with. Although laparotomies were performed upon eight of these patients, partial salpingectomy was done on three and bilateral salpingectomy upon five. Oophorectomy was performed on only one patient. I feel that the Elliott treatment did indeed limit the amount of surgery ordinarily performed upon such patients.

GROUP 4. POSTABORTAL PARAMETRITIS

This group consists of eight patients in whom there was a definite cellulitis, but no

abscess formation in the tissues immediately adjacent to the isthmus-uteri. Seven of these became sympton free, and I believe the other would have had she not unfortunately stopped treatment so soon.

GROUP 5. POSTPARTAL PARAMETRITIS

This condition occurred in eight patients who had had normal deliveries. These cases were mild, yet there was tenderness in the parametrium, a mild cellulitis. Six responded well to the treatments while results in two cases were fair.

GROUP 6. ENDOCERVICITIS

There were seven cases of endocervicitis, three with erosion, one without, and three cases that had had trachelorraphies with subsequent mild parametritis, were treated.

Although the eroded areas did seem to improve, I have always obtained such good results with electroconization or electrocauterization in one treatment that when I saw they were responding so slowly, I treated all seven patients with one of the latter technics with good results.

In the three women who had had trachelorraphies, there seemed so much resorption of scar tissue, with Elliott temperature over 120°, that I discontinued this type of treatment.

GROUP 7. VAGINITIS—SENILE

Three patients, so far, have received Elliott treatments, plus theelol. Two responded well, and one only fair. This patient is, however, still under treatment. Due to the marked inflammation and narrowing of the vaginas and decreased circulation in these cases, the temperature of the water, pressure, and duration of treatments were considerably less than in the other groups.

GROUP 8. POSTOPERATIVE INFLAMMATION

I performed bilateral salpingectomies upon four patients with chronic inflammation and after they were up and about, they experienced a little tenderness in the parametrium. Three were symptomatically relieved while the other one was practically so.

CONCLUSIONS

1. Although this series of cases is very small, too small in fact, to draw conclusions from, it does exhibit differences from other reports in literature, most all of which have been carried out in hospitals.

2. A striking feature is that the patients in Groups 1 and 2 became symptom free with an uneventful convalescence and did not require major surgery. This would indicate that if more of these patients could be treated in the manner outlined, in the acute and subacute stages, very little major surgery would be necessary at a later date, when the cases would perhaps become chronic.

3. Another very important point is demonstrated when one considers the age incidence in this series and the former frequent necessity of performing bilateral salpingectomy, or bilateral salpingo-oophorectomy resulting in the unfortunate picture of "artificial premature menopause."

4. Refractory cases to conservative treatment are found largely in the chronic group; these are rendered more amenable to surgery, limits the extent of surgery, thereby effecting better prognosis.

5. Postabortal, postpartal, and postoperative parametritis responded well to this type of therapy.

6. When surgery is necessary, "conservatism" should always be foremost in the mind of the operator.

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THE SERUM VOLUME TEST FOR THE HEMORRHAGIC DIATHESIS IN JAUNDICE: FURTHER OBSERVATIONS*

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Since only a small proportion of jaundiced patients exhibit a hemorrhagic diathesis, the problem at first glance might seem of no great concern to the surgeon. But two considerations promptly disturb any possible complacency. The first is that carefully compiled statistics attribute from 40 to 50 per cent of the death rate in surgical jaundice to this single cause. The second is that until very recently it was impossible to identify the patients who were likely to bleed by any method other than the one pessimistically suggested by Ivy, of waiting to see which ones did bleed.

There is still much in the problem of the hemorrhagic diathesis in jaundice that calls for elucidation, but within the last two or three years there have been a few very hopeful developments. The cause of the bleeding has been at least partially demonstrated. A few tests have been developed for the accurate identification of the patients who present this diathesis. And finally, with the demonstration of the cause of the bleeding, methods of preparation have been developed which are no longer empirical.

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CAUSE OF HEMORRHAGE

Because the three problems are so closely related, it seems well to begin this paper, which is devoted to the diagnosis of the hemorrhagic tendency, with a brief discussion of the underlying cause of the bleeding, and to close it with a brief discussion of the methods of preparing such patients for operation. We should say at the outset, too, as a matter of simple justice, that to the work of a single man and his group of co-workers we owe much of the light that has been shed upon this perplexing problem. That man is A. J. Quick, formerly of the College of Physicians and Surgeons of Columbia University, now of the Marquette University School of Medicine. To grant him his meed of praise detracts nothing from the work of others, much of which he has brilliantly applied to his own work.

Quick's excellent summary of our present knowledge of the bleeding tendency can profitably be used in this brief discussion. Four agents, he points out, are necessary for the clotting of blood. Prothrombin plus thromboplastin plus calcium results in an active enzyme, thrombin, which in turn reacts with fibrinogen to form fibrin, the basic factor in the blood clot. Repeated studies have never revealed any deficiency in calcium or in fibrinogen, and they may be eliminated from the discussion. The onus of the responsibility clearly lies upon one of the other two factors, but, until Quick himself developed the method, no test was available to demonstrate wherein the deficiency lay, and of what it consisted. Quick's test is based on the observation that the clotting time of oxalated plasma, when mixed with an excess of thromboplastin, which is readily available in the rabbit's brain, and with an optimum amount of calcium, can be employed as a direct measure of the prothrombin content of the plasma. In other words, if thromboplastin and calcium are kept constant, the rate of coagulation is necessarily dependent upon the concentration of prothrombin.

By the use of this test, Quick has been able to show a remarkably constant concen-

tration of prothrombin in normal blood, as well as in the blood of most jaundiced patients, although he noted a definite and sometimes a drastic reduction in the blood of a small number of the latter group. These are the patients with a bleeding tendency, and that tendency can be experimentally reproduced in one of three ways. Almquist and Stockstad, Dam and his associates, and Quick himself have shown that these three causes are a deficiency in vitamin K (the coagulation vitamin of Dam), sweet clover disease, and parenchymatous liver damage. Time does not permit a consideration of the exceedingly ingenious and interesting experiments which form the basis for these conclusions, but one striking observation must be mentioned, the very wide margin of safety in the prothrombin factor, which, Quick has shown, can be reduced to 15 per cent before a critical level is reached and which is associated with no change in the coagulation time until it has fallen to 20 per cent.

The quantitative prothrombin test devised by Quick is undoubtedly the only scientific diagnostic and prognostic test available today to demonstrate the bleeding tendency. Patients studied by it are thoroughly safeguarded, for it identifies those who exhibit this tendency, it permits the quantitative study of their improvement under proper therapy, and it indicates, finally, the safe period for surgery. But the test, regrettably, is not a simple test, in the sense that it requires the resources of a fairly well equipped laboratory and the services of a competent technician. It is not a bedside test, and it cannot be performed by a person untrained in laboratory methods. The surgeon who possesses these facilities has no excuse for not using it. The surgeon who does not possess them must continue his search for a simpler method.

Some months ago we published the description of a serum volume test for the bleeding tendency in jaundice, which has the virtue of simplicity and which in our hands has proved completely reliable. It requires for its performance only a syringe and a graduated test tube. An arbitrary amount of blood, preferably 3 c. c., is withdrawn and is allowed to stand at room temperature for four hours. At the end of that period the clot is removed and studied for firmness, retractility and other characteristics, and the serum volume in the tube is read. The index is determined by dividing the serum volume by half the volume of blood withdrawn, indices below 1, which is regarded as the standard of normal, being progressively indicative of a tendency to hemorrhage. Tests are repeated as necessary in patients who show a bleeding tendency, to check their improvement under preparation and to determine the safe time for operation.

We might repeat that this test was discovered accidentally, while we were studying jaundiced patients by the Grote test for sulphur compounds. We did not find it useful in this connection, although Carr, Foote and Naffziger have had more satisfactory results with it. Blood serum is necessary for its performance, and we happened to observe that an abnormally small amount was expressed from the clot of a patient who clinically showed the hemorrhagic tendency by frank purpura and hemorrhages from the mucous membranes. Thereafter we practiced quantitative studies of the serum volume in a series of non-jaundiced control patients whose blood studies were normal by standard methods, as well as in all our jaundiced patients. In the normal patients, and in most jaundiced patients, the serum volume was approximately half the volume of the blood withdrawn. In a few jaundiced patients, just as in the one who had furnished the clue, it was considerably less, though in all cases, under proper preoperative therapy, it could be elevated to normal or approximately normal.

Observation of the clot is an important part of this test, because a defective clot is the ultimate manifestation of a prothrombin deficiency. The immediate cause is a defect in fibrin, caused by an inadequate conversion of fibrinogen. Defective clots in patients with a bleeding tendency are large and bulky, they do not retract normally, they cover a large area of filter paper when they are removed from the test tube, and

they promptly fall to pieces when handled. The clot in normal subjects or in jaundiced subjects without a bleeding tendency is smaller, firmer and definitely retractile, and shows no tendency toward disintegration.

A routine blood count is necessary to demonstrate anemia, for which the proper correction must be made. If the corpuscular volume is reduced, as it is in anemic subjects, obviously the serum volume will be increased, the disparity increasing with the degree of anemia. Unless the proper correction is made, the index will be dangerously misleading, for it will naturally be higher than it really is. The corrected serum volume is arrived at by subtracting the red blood cells of the patient from five million red blood cells (the normal number), multiplying the result by half the blood volume withdrawn, and dividing the product by five million red blood cells. The index is then calculated as usual. We have also learned from experience that if the blood is not transferred promptly from the syringe to the tube, the clotting process will begin in the syringe and will be disturbed by the transfer, the resulting index being lower than it really is.

We reported in our first contribution 19 cases of obstructive and seven cases of non-obstructive jaundice studied by this test. In this contribution we are reporting 14 cases of obstructive and seven of non-obstructive jaundice which have been studied by this method, by the Ivy venous pressure bleeding test, and by the Quick hippuric acid test of liver function. This is not a statistical study. We are reporting these cases because they have been studied by these three methods, the combination of which has permitted certain significant conclusions.

In the first place, our results with the serum volume test continue to be entirely satisfactory. No patient has indicated any tendency to hemorrhage when the index was within normal limits, and the only ones who have exhibited such a tendency are the patients whom we expected to bleed. We shall comment upon them shortly.

The highest serum volume index at which a patient bled was 0.71, the lowest at which hemorrhage did not occur was 0.78. Apparently the lower limit of safety is somewhere between those two points. Until our experience is considerably wider, however, we personally shall not be willing to operate when the index is below 0.8, as an absolute minimum, and in view of the readiness with which such patients respond to preparation by glucose, transfusion and dietary measures, we think it the part of prudence to wait until the level is approximately normal.

The two patients who bled deserve a brief comment.

CASE REPORTS

The first, a white woman 46 years of age, had a simple exploration for carcinoma of the pancreas, which was too advanced to permit even a short-circuiting operation. By our personal standards her preparation was inadequate and her serum volume index of 0.71 below the safe minimum. The surgeon reported that there was some oozing at operation, and she lost blood through the drainage tube at intervals from the second to the eighth day. On the ninth postoperative day, after repeated transfusions and intensive glucose therapy, her serum volume index reached 0.83.

The second patient who bled was a white male, 69 years of age, who also had a carcinoma of the pancreas, for which cholecystgastrostomy was done. His serum volume index was 0.62 and his preparation was not adequate. The surgeon reported oozing at operation, and the index the day after operation was 0.66, practically what it was three days earlier. He died on the fifth day, from peritonitis, not from hemorrhage, but at autopsy the peritoneal cavity was full of bloody, bile-stained fluid, and the stomach contained coffee grounds material. A soft, waxy clot was seen at the line of the anastomosis.

These two cases suggest several lines of thought. One is that oozing is more usual than hemorrhage at operation, a phenomenon which can easily be explained by the defective character of the clot. The vessels are plugged, but they are not plugged adequately. Furthermore, as we have previously pointed out, bleeding tends to be capillary rather than arterial or venous, and is a treacherous variety which is likely to be entirely overlooked. Another point brought out in these cases is that bleeding is even more likely to occur after opera-

tion than at operation, because, as Ivy and his associates note, restlessness, vomiting and similar factors are likely to dislodge the defective clot.

Our results with the Ivy venous pressure bleeding time, as these cases show, have not been entirely satisfactory, though we use it routinely and shall continue to use it routinely. In the first patient just mentioned, the Ivy test with the tourniquet in place for four minutes was slightly above the upper limit of normal, and it was within the normal range the day after operation, which was the day before bleeding began. The Ivy test in the second patient was normal throughout. In a recent report by Boys, the Ivy test apparently demonstrated the patients who did bleed, but indicated a hemorrhagic tendency in some 65.4 per cent of the patients who did not bleed. The report furnishes no clue to this curious situation. Perhaps the length of time the tourniquet was kept in place had something to do with it. It is our custom to make two tests, one after the tourniquet has been in place for one minute, and another after it has been in place for four minutes. There is often a considerable variation in the two bleeding times, though the variation is not at all constant. We have noted also that this test tends to be less reliable as age increases, and presumably, as arteriosclerotic changes develop.

We have observed in the two patients who bled another point which is well worth discussing. We have always personally questioned the direct relationship between the state of the liver and the hemorrhagic tendency, and we still question it, in spite of the apparently conclusive case which Quick has reported in his latest communication on this subject. That a depressed liver function and the hemorrhagic tendency may coexist we do not deny, nor do we deny that they may improve together, as happened in Quick's case, but we still doubt the cause and effect relationship.

In the first of the cases we have reported the patient indicated a hemorrhagic tendency by the serum volume index, and proved it by bleeding, yet the Quick hippuric liver function test before operation was 2.175, 72.5 per cent of normal, which is a safe level, we have found. It fell progressively as her condition grew progressively worse, and as, presumably, the hepatic metastases seen at operation involved more and more of the liver tissue, yet no further hemorrhagic tendency was evident, and the serum volume index improved as the liver dysfunction increased. In the second case a marked hepatic dysfunction was present before operation (0.823, only 27.5 per cent of normal) in association with the serum volume index of 0.62. The liver function was sustained at approximately the same level until death by the use of large quantities of glucose; the serum volume index unfortunately was not repeated.

Another case reported in our first communication furnishes further disproof of the direct relationship between dysfunction and a bleeding tendency.

CASE REPORT

A white male, 69 years of age, was very deeply jaundiced from a common duct obstruction. The first serum volume index was 0.48; after intensive preparation it reached normal, 1. Liver function by the Quick test was 1.823, 60.8 per cent of normal, after intensive preparation; it had originally been 0.786, 26.2 per cent of normal. This patient exhibited no hemorrhagic tendency at operation, nor as autopsy showed, did he bleed thereafter, but he died a typical hyperpyrexic death as the result of his hepatic dysfunction.

A similar case is reported by the Mayo Clinic surgeons. Their patient had proved her hemorrhagic tendency by bleeding at a previous operation; she was adequately prepared and did not bleed at the second operation, but she lost her life from liver insufficiency. On the opposite side of the ledger, we have a large number of patients who exhibited a more or less marked depression of liver function, but whose serum volume index was normal and who did not bleed at operation.

PREPARATION FOR OPERATION

We have prepared these patients in various ways. Glucose is never omitted, because of its effect upon the liver, and we

have found Althausen's method of oral administration very satisfactory. We have made free use of transfusion, the best of the immediate methods, according to Quick, because it replaces the missing prothrombin. Transfusion also increases the oxygen saturation curve of the blood, as Judd and Hawkins have pointed out, and so corrects the anoxemia which is sometimes marked in jaundiced patients, particularly when there is associated liver damage. It must be remembered, however, that transfusion has only a temporary effect. It produces no permanent improvement, and preoperative transfusion should never be regarded as a safeguard against postoperative hemorrhage. Walters' statement that there is an optimum time for operation after transfusion is now clear in the light of Quick's demonstration that its good results are due to the replacement of prothrombin.

A high carbohydrate diet is always indicated in such cases, and since the work of Quick and of the Mayo Clinic surgeons has been published, we have supplied vitamin K in the form of tomatoes, lettuce and cabbage, to correct the vitamin deficiency which in the light of present knowledge seems to be the basic deficiency. Bile salts are administered also, for vitamin K is not absorbed in their absence when the obstruction is complete, as the studies of Schmidt and Greaves have shown. We have prepared three patients with initial low serum volume indices by this method, raising the index to normal before operation and carrying the patients through operation and convalescence with no bleeding whatsoever. We also prepared another patient, whose initial serum index of 0.48 rose in ten days to 0.65. After the initial rise in the serum volume index, however, this patient failed to improve. She was not cooperative, but even with transfusion the serum volume index did not rise above 0.70, and remained at that level only temporarily. At the end of four weeks, in desperation, she was operated on, though her serum volume index was only 0.55. Exploration and biopsy were done for a condition which was diagnosed as malignancy of the biliary tract at operation and proved to be carcinoma of the gallbladder, with metastases to the liver and mesentery, at autopsy. The patient oozed very freely from the incision and the biopsy site at operation, and bled to death on the seventh postoperative day. Autopsy revealed about two liters of free blood in the peritoneal cavity, and many organized blood clots adherent to the intra-abdominal structures.

The excellent results reported by Osterberg and Snell, by Quick, and by others who are using vitamin K in its concentrated, readily assimilable form, which is not available to us, plus the good results we personally have secured by our cruder methods of administration, furnish excellent proof that this factor will correct the basic deficiency responsible for the bleeding tendency.

Since this paper was written, two other cases have been observed in which the serum volume test showed a definite tendency to hemorrhage, preparation for surgery was not satisfactory, and postmortem examination confirmed the bleeding tendency.

SUMMARY

1. The underlying causes of the hemorrhagic diathesis in jaundice are discussed, with special reference to Quick's prothrombin theory.

2. The serum volume test for the hemorrhagic diathesis in jaundice, which we described in 1937, is described again, and additional details for its use are mentioned.

3. Twenty-one cases of jaundice are reported, all of which were studied by the serum volume test, the Ivy venous pressure test, and the Quick hippuric acid test of liver function.

a. The serum volume test proved correct in all cases, but the Ivy test did not.

b. The frequent lack of correlation between the serum volume test and the Quick hippuric acid test introduces the question of whether depressed liver function can be invoked to explain all cases of bleeding in jaundice.

4. The preparation of jaundiced patients for surgery is outlined with special reference to the use of transfusion, glucose, and vitamin K.

REPRESENTATIVE CASES OF OBSTRUCTIVE JAUNDICE STUDIED BY SERUM VOLUME INDEX, IVY BLEEDING TIME, AND QUICK LIVER FUNCTION TEST

Case No.	Red Blood Cells	Platelets	Icteric Index	Clotting Time	1 Min. Ivy Time	4 Min. Ivy Time	Serum Volume Index*	% Normal-Quick Test**	Diagnosis and Procedure	Comment
1	4,470,000	147,000	50	180 sec.	105 sec.		.65	90.4	Choledocholithiasis	Prepared by vitamin K and bile salts. Note elevation of liver function by liberal use of glucose.
	3,860,000		100				.71	101.0	Choledochostomy	
	3,390,000		200				.78	119.0		
							.93		Stone in ampulla	
2	4,180,000	111,000	85	720 sec.	135 sec.		1.00	95.6	Transduodenal resection	
								95.4		
								85.0		
3	4,870,000	303,000	65	540 sec.	60 sec.	30 sec.	.83	85.7	Choledocholithiasis	Prepared by vitamin K and bile salts. Note post-operative drop in liver function.
			25					54.4	Choledochostomy	
								104.3		
4	4,360,000	370,000	65		105 sec.	225 sec.	.85	97.0	Cholelithiasis	
								89.5	Marked pancreatitis	
								70.0	Cholecystostomy	
5	4,340,000	150,000	100	270 sec.	60 sec.	105 sec.	.76	40.4	Carcinoma of pancreas	Prepared by transfusion, vitamin K and bile salts. Note low liver function and relatively high S. V. I.
			166					63.0	Cholecystgastrostomy	
							.90	92-87.4		
6	4,440,000	190,000	33	45 sec.	60 sec.	90 sec.	.60	112.1	Choledocholithiasis	Prepared by vitamin K and bile salts. Note low S. V. I. and high liver function.
	4,320,000						.62	41.2	Choledochostomy	
							1.00	81.4		
7	4,620,000	320,000	30	240 sec.	150 sec.	180 sec.	.98	79.5	Cholelithiasis	
							.87	87.0	Cholecystectomy	
								120.0		
8	4,410,000	150,000	65		71 sec.	255 sec.	.75	76.0	Common duct stricture	
			30		40 sec.	75 sec.	.78	92.5-122	Cholecystgastrostomy	
								72.8		
9	4,040,000	380,000	50	240 sec.	180 sec.		1.06	127.6	Cholecystitis, hepatitis	
			25					56.4	Cholecystostomy	
								66.5		
10	3,570,000	320,000	50	630 sec.	225 sec.	150 sec.	.71	72.5	Carcinoma of pancreas	Bled intermittently one week postoperative. Second S. V. I. after transfusion, eighth postoperative day. Remained in hospital until death. Note decrease in liver function.
	4,520,000	390,000			210 sec.		.83	69.2	Exploration	
								44.8		
11	3,550,000	270,000		450 sec.	270 sec.	90 sec.	.62	27.4	Carcinoma of pancreas	Oozed at operation. Evidence of further bleeding at autopsy. Second S. V. I. second day.
	2,410,000	200,000		645 sec.	105 sec.	180 sec.	.66	28.8	Cholecystgastrostomy	
								27.0		

*Serial test.

**First test before operation, second test first or second days after operation. Final tests on discharge.

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DISCUSSION

Dr. George M. Decherd, Jr. (New Orleans): Assay of all conceivably important factors has until recently failed to give any hint as to the nature of the hemorrhagic tendency in certain cases with jaundice. Evidence adduced in the past few years that this bleeding is correlated with, or likely due to, a deficiency in the blood prothrombin content, represents the first real contribution to our knowledge of this serious complication. Used in conjunction with other information obtained from the experimental production of prothrombin deficiency, it implies the proper and adequate prophylactic and therapeutic measures to be employed in these cases. An inadequate intake of vitamin K, hepatic damage, and the feeding of spoiled sweet clover all appear to lead to a bleeding tendency by reducing the content of prothrombin in the blood.

While not conclusive, the evidence seems strong that prothrombin, as well as fibrinogen, is manufactured in the liver. Certainly both may be reduced in severe liver damage, though the level of the former is more readily depressed. The functions of the liver are so varied that it is scarcely to be expected that evaluation of one will make possible a prediction of the status of all of the others. Hence, it is not surprising that the level of blood prothrombin, if it be a product of the liver, does not always run parallel with the function of the liver as determined by tests that have proved of practical value for other purposes.

Vitamin K is fat soluble, but is not a sterol, and does not occur in sources rich in vitamins D or E. It is present in human intestinal contents, either ingested in food or possibly produced by the action of intestinal bacteria. The point to be emphasized is that its absorption requires the presence of bile salts, and is hence deficient in complete biliary obstruction. When such circumstances have led to a reduction of blood prothrombin to 15 to 20 per cent of normal, the bleeding tendency will become manifest, and for administration of vitamin K to be efficacious, it must be supplemented by the use of bile salts or whole bile by mouth or tube. This mode of therapy has proved successful elsewhere in reducing abnormally prolonged prothrombin times in such patients.

Reports available at present indicate that prolongation of the prothrombin time is a valid basis for the prediction of postoperative bleeding in jaundiced individuals. However, facilities for this determination are not widely available, for although it is not extremely difficult to perform, the reagents and conditions under which it is done must be rigidly controlled. Determination of the serum volume index is not open to these objections, and on the contrary may easily and conveniently be done. If further experience in its use confirms the present well-founded opinion as to its utility, it will be recognized as a procedure of great practical value, and render its author deserving of the highest commendation.

Dr. Leo N. Elson (New Orleans): I want to bring out another angle from which I see the causation of hemorrhagic diathesis in jaundice, not that I want to criticize Dr. Boyce's valuable paper and the important test therein described, but to throw more light and give more emphasis to the right treatment, or at least leading to a treatment of some consequence.

From physiologic studies we know that bile is not only an excretion product but has many indispensable enzymotic functions. Among others, it is the most important enzyme in the digestion and absorption of fats and fatty acids, in saponifying them and combining them with calcium, potassium and sodium as oleates or soaps and as such they are absorbed by the body.

Next, bile is well known to have a function of lowering the coagulability and viscosity of the blood. Some years ago, I wrote a monograph on the treatment and prevention of coronary thrombosis. In it, I recommended, among other things, the use of bile and bile salts in all thrombotic states of the blood and hypertensive diseases. How does bile act? By saponification and fixing the available blood calcium to the lipoid substances and cholesterine bodies in the blood. By this means

it lowers the coagulability and viscosity of the blood, as fibrinogen minus calcium will never form fibrin.

In jaundice, when the blood contains excessive bile, the calcium is constantly being fixed to the lipins and its availability to combine with fibrinogen to form fibrin, the essential part of the clot, is diminished; hence the hemorrhagic diathesis in jaundice.

For the treatment, Dr. Boyce spoke about the use of vitamins, and the use of calcium was also mentioned. I want to add with emphasis that not only calcium in full sized doses should be used orally and parenterally, but also parathyroid extract should be prescribed to produce a superabundance of available calcium in the blood, and thus lessen the danger of or abolish completely the hemorrhagic diathesis in the jaundiced patient.

THE CHRONICITY OF PULMONARY TUBERCULOSIS*

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Pulmonary tuberculosis is generally accepted as a debilitating and relapsing disease, but its chronicity, like so many other concepts which are clinically taken for granted, is not appreciated as thoroughly as it might be. If it were, some of the tragedies of the disease could be prevented, and practitioners in general might not be so apathetic to the problems entailed. It seems strange and perhaps ludicrous to be required to emphasize today the fact that tuberculosis is frequently fatal, tends to last much longer than one usually anticipates and is ever with us; yet, the complacency with which many practitioners view the disease permits only the conclusion that they have forgotten how long drawn out and expensive phthisis pulmonis may be. It is true that there is a declining mortality rate, but if we are to make that rate fall more yet, we must appreciate that our task is indeed an arduous one.

Tuberculosis is a costly disease, much more so than any other communicable malady. In a series of 100 well studied cases, Sawyer and Richard[1] found that before the average patient was restored to working condition, he had lost more than two and a quarter years, and that the cost of rehabilitating him was $4,602.00. These costs included not only the actual money spent for medical care but also for the auxiliary measures and for the time lost from employment. The average cost per case varied with the extent of involvement: thus, the minimal case might cost $3,218.00 and the moderately advanced $5,578.00, while the far advanced cost a little less, $4,061.00, presumably because most of the patients with far advanced tuberculosis do not live long enough to be more expensive. These costs are not always predictable on the basis of extent of involvement; one of the minimal cases cost $24,509.00 before being rehabilitated.

Now, it is quite possible that many of the poor results of treatment ensue because the marked chronicity and costliness have not been sufficiently impressed on practitioners. It is all too common to find that patients are advised simply "to take it easy" once a diagnosis of pulmonary tuberculosis has been rendered. Admittedly, it is a difficult thing to convince a patient that his only hope of regaining economic usefulness lies in absenting himself from work over a period of months or years; yet, unless this is done at the beginning of treatment, the patient will eventually lose far more time and money as a result.

Certain facts have tended to obscure the natural history of the disease. When the "rest cure" was first instituted, it was found that if tuberculous patients were allowed continuous rest in bed for a few months, a great change usually occurred. They gained weight, the temperature and pulse became normal, and all symptoms disappeared. This rather startling metamorphosis caused clinicians to believe that pulmonary tuberculosis was easily cured, and after a short period of treatment, they considered such patients "arrested." The introduction of the roentgen ray, however, demonstrated conclusively that although clinical symptoms may disappear rapidly, the tuberculous process in the lung often clears and heals very slowly and usually

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only after years of prolonged and continuous bed rest. Unfortunately, such facts are not sufficiently appreciated even today, after years of re-emphasis placed on them. It is still too common an occurrence to find patients who have taken several "cures" only to relapse into a more serious and progressive phase of the disease. Following a period of therapy, the improved state of well-being has led patients and physicians to conclude that stay of the disease had been obtained and that restrictions might be relaxed. Only the bitter experience of relapses has served to indicate the fallacy of such reasoning.

It may be that in the majority of instances, the patient voluntarily ceases treatment when he feels well enough to resume work. It should be recalled that someone has wisely stated that "The medical errors of one generation are the popular beliefs of the next"; patients who today want to terminate their course of treatment when symptoms cease are unconsciously applying the lessons learned from an earlier generation of medical counselors. While this is true, the fact remains that many patients are told simply to "take it easy" even today; and if reliance is placed on symptoms only, many patients will be dismissed much too soon.

MORTALITY RATES

Because there has been a steadily decreasing mortality from tuberculosis, we have been lulled into a false sense of security and have been led to believe that the problem of tuberculosis is well under control. The shocking complacency of many practitioners toward tuberculosis is all the more unfounded in light of studies of the present status. Drolet[2] has studied the figures of the past twenty years and has shown conclusively that despite the marked decrease in morbidity and mortality rates there has been practically no decrease in the case-fatality rates for tuberculosis. This is to say that today there is less chance of becoming infected with tuberculosis, and fewer people are dying of the disease, but that the patient who develops it has as much chance of a fatal issue as in years gone by. This horrifying revelation is well documented. Drolet has concluded that ". . . . sanatorium or surgical treatment of pulmonary tuberculosis would seem so far to have had little effect upon the case-fatality rates of the entire tuberculous population in the communities studied, and segregation of active cases along with other general sanitary measures and the improvement in living and working conditions has undoubtedly been a powerful factor limiting infection and preventing the spread of tuberculosis."

Hilleboe[3] has compared the mortality rates of patients discharged from tuberculosis sanatoria with those of similar groups in the general population and has shown that for the patients in his series with minimal tuberculosis the risk of fatality is four times that of the general population; with moderately advanced tuberculosis it is sixteen times and with far advanced forty times. He shows that where a diagnosis is made in the early or minimal stage the risk of fatality is considerably less than where the diagnosis is not established until the process is advanced.

The clinical maxim, "Once infected, always infected" finds corroboration in the necropsy studies of Robertson[4] at the Mayo Clinic who reported a series of routine autopsies where the diagnosis of tuberculosis was never suspected antemortem and found that tuberculous infections may occur and pursue their course without once attracting the attention of the physician or the patient; that recognized tuberculous infections may subside and be regarded throughout life as healed yet still remain continuously active; and that apparently healed tuberculous lesions may become clinically active after varying intervals.

Such studies are certainly not in keeping with the lack of interest encountered these days. In commenting on the general problem of tuberculosis, Varrier-Jones[5] remarked that "The fact that a survey of the tuberculosis problem over the past fifty years shows a decline in the death—and incidence—rates is instructive, but is likely to breed complacency. Action instead of complacency is called for in a disease that

is killing four times as rapidly as traffic and for which people fear treatment more than the disease. We look for early cases and discover the cases found are not early. We seem to be still fascinated by clinical manifestations of disease and make efforts to study the pre-breakdown or really early stage. Possibly, our definition of an early case is not correct, and we must strive to antedate our conception of onset."

Now, it ought to be realized that such an uncomplimentary survey does not mean that the entire tuberculosis situation is gloomy. The methods of treatment at our disposal are efficacious and productive of good end results as many studies testify. The above is by no means an indictment of our present forms of therapeusis; rather it is a plea for their more vigorous and consistent use. Patients can be rescued from the ravages of tuberculosis, but only when treatment is intelligently planned and courageously maintained. The practitioner may use the so-called hygienic-dietetic or one of the several forms of collapse therapy or any combination of these, but he must use them early and thoroughly enough. Unless he appreciates that "Tuberculosis never seems as serious as it is" as Head[6] has so wisely remarked, he is apt not to apply proper measures of control; and cases of tuberculosis will lapse needlessly into the chronic stage, the stage where often only heroic measures will suffice, if these avail anything.

THE EARLY DIAGNOSIS

It is virtually axiomatic that to prevent the ill effects of neglected tuberculosis, the disease must be recognized when early or minimal. This is not an easy thing to do most of the time, and there is no denying the fact that it imposes quite a burden on the physician. The first requirement is that the practitioner shall become "tuberculosis-minded" and shall keep phthisis ever in mind. He must always remember that tuberculosis is a possibility, nay a probability, in any state of ill-health when the cause cannot at once be found. When it will be borne in mind that this disease in its earliest stages is seldom ushered in with characteristic symptoms and that typical physical signs are not often present, there will be more recourse to sputum examination and x-ray filming; and when this time comes there will be more diagnosis of early tuberculosis, the kind that the practitioner wants to treat but seldom sees.

Only too often is he deceived into thinking that tuberculosis does not exist because the patient has no cough or expectoration, is gaining weight instead of losing and has no fever. Time and again during the course of routine case-finding studies, patients with minimal or moderately advanced lesions have been discovered, and they had few or no symptoms at all. There is one way and only one way to eliminate tuberculosis as the probable cause for a patient's symptoms and that is to show a normal roentgenogram of the chest. To be sure roentgenography has its limitations, but its assistance has been invaluable for determining the presence and extent of thoracic lesions. The fact remains that if ever minimal tuberculosis is to be detected by intent and not by accident, the disease must be considered where there is nothing in the patient's appearance to suggest it and when even those "in the picture of health" are held suspect until proved otherwise.

It is true that many patients conceal or ignore symptoms of early disease and therefore do not report until their lesions have advanced, and it is also true that many patients appear to have gone almost into the depth of advanced tuberculosis without the preliminaries of a minimal stage. Perhaps such cases are more frequently encountered than is generally realized, but here again only the acuity of the alert practitioner will serve to bring these patients to proper treatment at a time when physical and economic salvage may yet be accomplished.

FUNDAMENTALS OF TREATMENT

Whether the disease is minimal, moderately or far advanced, the principles of treatment are the same; it is only the intensity and their mechanical applications which differ. Generally speaking, the

more prolonged is the disease, the more vigorous and the more expensive must treatment be. There is, of course, no way of determining at the onset whether a lesion will progress, retrogress or remain stationary; therefore, only repeated studies and constant observation will suffice. Failure to recognize this will result only in disaster. It is far safer to err on the side of caution and to consider the beginning lesion as grave than to make the mistake of minimizing a process and to fail to render consistent treatment.

It is obvious that only by study of the serial roentgenogram can the practitioner properly follow the course of the disease. Studies of the white blood cell formula and of the red cell sedimentation time will assist in determining the degree of activity of the lesion, yet the film is essential for an appreciation of the changing anatomic-pathologic state. It is particularly important to see whether the lesion is responding to proper methods of therapy. The greater majority of patients will continue to have a modification of the hygienic-dietetic treatment at the onset whether this be instituted in the home or in a sanatorium. If this seems not to be efficacious after a period of time, it is advisable to institute surgical measures for compression of the lesion. Especially is such a plan imperative where there are cavities. Barnes and Barnes[7] have declared that in chronic fibrocaseous tuberculosis with cavitation and sputum containing acid-fast bacilli, the prognosis is very gloomy, for 95 per cent of such patients are dead at the end of five years' time. While such figures are considerably higher than those of other authors, it is definitely known[8] that the huge majority of patients whose cavities do not become completely closed and whose tubercle bacilli do not completely disappear will suffer a replase in the near or distant future and die of tuberculosis or its consequences. The most conservative figure is that 50-75 per cent of them will be dead at the end of this five year period. A certain percentage of these cavities will undergo spontaneous healing if optimal conditions of rest are maintained, but the time of closure is much longer without surgical collapse than with it. It is therefore a safe rule that whenever cavities cease to diminish in size or begin to increase, they are by all tokens ready for surgical measures. It should not be forgotten that the patient with one or more cavities is not only a menace to himself in that spread of the disease is an ever-present threat, but is also a menace to others. His sputum is always loaded with tubercle bacilli, and he is accordingly the most prolific disseminator of the disease. The cavity that does not very soon close of its own accord is one of the most potent stimulators of the chronicity of tuberculosis and tends to perpetuate the disease inside and outside the patient containing it. It must be appreciated that the course of this cavity can be followed by studying x-ray films taken at approximate intervals as well as by studies of sputum and blood. We are helped exceedingly little, if at all, in the average case by a recital of the patient's symptoms or elicitation of physical signs. It is by no means a rarity to see a cavity continue to grow in a patient whose symptoms have long since disappeared and who radiates the appearance of good health, even whose well-being is apparently beyond question. It is common observation that symptoms appear only during periods of exacerbation and sometimes not even then; and their presence may be a warning of imminent or future danger, but their absence certainly does not afford us much of a guide.

IMMOBILIZATION THERAPY

There is abundant statistical evidence to show that collapse therapy offers much to the patient with chronic tuberculosis. If his disease is still minimal and fails to respond to conservative therapy, artificial pneumothorax is the method of choice. Turner and Collins[9] have shown that when combined with rest treatment, this procedure yields excellent results. Even in the moderately advanced and the far advanced patients, it will be followed by some benefits, although its value diminishes, generally speaking, as the amount of disease increases. Those for whom artificial pneu-

mothorax cannot be applied because of technical reasons may be helped by phrenic nerve operations and by thoracoplasty. The most important thing is not which operation shall be employed (because each procedure is reserved for the type of patient who stands the most to benefit by it), but the really significant thing is that some form of collapse therapy shall be used before it is too late. Alexander[8] has justly compared the surgery of tuberculosis with appendectomy for acute appendicitis. Operation for either disease is not radical; it is conservative in the sense that it saves tissue and prevents further loss of function and possibly of life.

The patient with chronic tuberculosis[8] runs great risks if his process cannot be controlled by bed rest alone. The insidious or sudden progression of tuberculous lesions in one or both lungs, the formation or increase in size of cavities, the increasing rigidity of a diseased lung from scar tissue, the development of pleural adhesions, the dangers of occurrence of the various complications of pulmonary tuberculosis at any stage and the harmful effects of prolonged tuberculous toxemia are the risks run from failure to check an active tuberculous lesion as soon as possible. A large number of patients have died from tuberculosis when, at the time they were first seen by the physician, they were suitable for some form of collapse therapy.

If, then, the disasters of relapse and progression of lesions are to be prevented, all patients must be treated intensively and thoroughly. It is necessary to lean heavily on radiologic methods as the basis for determining progression or retrogression of the disease. It is well established that the more intensively the patient has been treated and the longer the period of inactivity allowed for the tuberculous process to become quiescent, the less the chances are that relapse will occur. Fales and Beaudet[10] have attracted attention to the fact that the nearer a patient's condition approaches the status of what could be called radiologically healed lesion, the fewer the post-hospitalization reactivations and deaths there will be. If this is carried to its logical conclusion and patients are not dismissed from treatment until there is complete radiologic evidence of a healed lesion, less than 1 per cent of all patients will relapse. Failure to follow this rule and insistence on being guided by the patient's symptoms or state of well-being is courting the disaster of fatality and is unquestionably contributing handsomely to the chronicity of tuberculosis.

SUMMARY

The chronicity of pulmonary tuberculosis is not appreciated as widely as it should be. Because of this and because practitioners have paid more attention to the symptomatology of patients than to their radiologic aspects, relapses in tuberculosis are frequent. It should be emphasized that pulmonary tuberculosis is an exceedingly chronic disease and that at the onset it is not possible to state whether the disease will be active a period of months or years or even whether a good result can be foretold. The only logical approach to the problem is to make every effort to diagnose the disease early, at the time when it is most amenable to treatment; failing this, to treat all cases intensively and thoroughly and not to reserve surgery for the hopeless or the moribund patient but to utilize surgical principles for all patients who do not show prompt and marked response to the hygienic-dietetic regimen, and to consider each patient as having active pulmonary tuberculosis until there is definite, persistent and consistent radiologic evidence of arrest of the process.

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ADVANTAGES AND DISADVANTAGES OF CYCLOPROPANE ANESTHESIA*

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Cyclopropane, a hydrocarbon gas with the chemical formula C_3H_6, was first described by Freund in 1882. It was introduced by Henderson and Lucas of Toronto[1] as an experimental anesthetic gas in 1929. Ralph Waters of Wisconsin[3,5] first used the gas on humans in 1930. Since that time it has been widely used by anesthetists in America and on the Continent and has steadily gained popularity. This gas is one and one-half times as heavy as air, has a somewhat sweetish, pungent odor, but is not unpleasant when administered properly. It produces anesthesia in relatively low concentrations,[3] 7.4 per cent for first plane anesthesia to 43 per cent for respiratory arrest.

I do not laud cyclopropane as the ideal anesthetic, but wish to call attention to its advantages as well as disadvantages. It must be remembered that each agent has its own definite use and limitations, and, if these limitations are known and adhered to, cyclopropane approaches the ideal inhalation anesthetic and demands a definite place in the armamentarium of the physician-anesthetist.

DISADVANTAGES

1. Cyclopropane is an expensive gas, being about twenty times more costly than ethylene and nitrous oxide. Because of this, expensive apparatus, consisting of a machine with a closed air-tight carbon dioxide absorption system, must be used for administration.

2. Because of its potency, an exacting technic must be used which cannot be mastered by the physician who gives an occasional anesthetic.

3. The usual signs and symptoms of anesthesia are of little value. Color cannot be relied upon because the patient is always pink up to the time of respiratory arrest. Eyeball movement is more reliable but still variable. The patient may be well relaxed with all abdominal and laryngeal reflexes absent with the eyeball still roving or off center, and viceversa. The most reliable sign, but difficult to appreciate, is the breathing. It is always shallow because the carbon dioxide is absorbed and cyclopropane is a mild respiratory depressant; there is no increased depth of respiration when first plane is reached as with the other agents. With cyclopropane the respiration becomes progressively more shallow until respiratory arrest occurs. Therefore the depth of anesthesia can be judged by the depth of respiration, but the changes are minute.

4. Cyclopropane being a respiratory depressant, respiration may cease while the patient is of a good pink color and before relaxation has occurred.

5. There is an increase in blood pressure during induction which gradually approaches normal as the anesthetic continues in an even plane. This would contraindicate its use in extreme hypertension.

6. There may be increased bleeding in the wound, but there is no significant change in the coagulation time.[3] It is possible that the elevated blood pressure may cause more capillary ooze.

7. The gas is inflammable in concentrations ranging from 3.8 per cent to 50 per cent.[1,5] However, it is always used in a closed system so that the only way an explosion could occur would be by production of a spark in the system. This is impossible as the relative humidity in this system is 100 per cent as soon as the patient takes a few breaths.

8. With too high concentrations of the gas there may be arrhythmia or slowing of the pulse and at times tachycardia. With

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increase of oxygen content of the mixture the pulse invariably returns to normal. Electrocardiographic studies have shown that there is no change in the heart function until one and one-half times the concentration necessary for respiratory arrest has been attained, provided proper aeration of the lungs is maintained.[19] If anoxia occurs arrhythmia develops which ceases when the blood is oxygenated.

9. During long operations, which are shocking because of their length and loss of blood, cyclopropane seems to bolster the circulation in an occasional case. As long as the anesthetic continues the pulse is slow and the blood pressure good. But soon after anesthesia ceases the blood pressure falls and the pulse rate becomes elevated, indicating some degree of shock. This state of affairs has given the surgeon and anesthetist a sense of false security, both thinking the patient's condition to be excellent as the operation nears completion, only to find some degree of postoperative shock. I have no explanation for this except that possibly the organism has become accustomed to the high oxygen concentration in the anesthetic mixture and does not do well when suddenly put in a normal atmosphere with 21 per cent oxygen.

10. Circulatory complications are slightly more frequent with cyclopropane than the other anesthetic agents,[12] but in all reports the majority of bad risk cases were anesthetized with cyclopropane.

11. The degree of muscular relaxation equaling that of deep ether or spinal anesthanalgesia cannot be obtained at all times. There is usually some muscle tone present, and the surgeons who are used to deep ether or spinal analgesia often find it difficult to operate on the patient anesthetized with cyclopropane. However, the surgeons who are used to straight nitrous oxide or ethylene extol the marvelous relaxing power of cyclopropane.

The persistent muscle tone mentioned above may not be a disadvantage, as anesthetic shock occurs on the deeply anesthetized patient when all muscle tone including that of blood vessels is lost. Thus, with cyclopropane, anesthetic shock is seldom encountered.

11. A copious flow of saliva occurs in about one-fifth of the cases. This is not due to local irritation of the gas, but is a central effect and is limited to the mouth and pharynx. This saliva may come in contact with the vocal cords and cause laryngospasm during the anesthetic. Gentle pressure on the breathing bag usually prevents the spasm if instituted at the first evidence of crowing.

12. There is one pulmonary complication which is more frequent after cyclopropane than the other agents, namely atelectasis of the peripheral alveoli. This does not occur in short anesthetics, but is more apt to happen in the longer cases. The reason for this is that during a long case the bag may have to be refilled several times because of a slight leak, or the gases may be lost as the mask is adjusted from time to time. After so long a time there remains only cyclopropane, oxygen, and a small amount of carbon dioxide. The inert nitrogen which was in the lungs and trachea at the beginning of the anesthetic has been reduced to an infinitesimal amount. The remaining gases, cyclopropane, oxygen, and carbon dioxide are absorbed rapidly by the lungs. Because of the abundance of oxygen, lack of respiratory stimulation by the low carbon dioxide concentration, and the depressant effect of cyclopropane, the breathing is very shallow. The peripheral alveoli may have no movement; thus these gases are absorbed relatively quickly and peripheral atelectasis results. This complication can be obviated by using some inert gas, which is absorbed slowly, or by hyperventilation at the end of the anesthetic. Both of these things are done when the anesthetic is unduly long. Helium, which is an inert gas and is absorbed seventeen times more slowly than oxygen, is added to the mixture to replace the inert nitrogen which has been lost. Also, after every long operation the patient is hyperventilated by blowing a stream of carbon dioxide on the face until several deep breaths have been taken. This insures expansion of the peripheral al-

veoli with air. Relatively few postoperative pulmonary complications will be encountered if these procedures are routinely carried out.

ADVANTAGES

1. Induction is fast, easy, and pleasant with no throbbing or choking. Deep anesthesia is reached in from five to six minutes, about half the induction time of ether. There is seldom any excitement stage, as this is passed over quickly and the patient senses no feeling of suffocation or irritation to the mucous membranes. Recovery time is short, five to twenty minutes, depending on the depth and length of anesthesia.

2. An abundance of oxygen is present at all times, as fourth stage anesthesia can be reached with 43 per cent cyclopropane.[3] The oxygen content always exceeds 20 per cent and with the carbon dioxide absorption technic the maximum carbon dioxide concentration is 4 per cent.[3] These factors assure easy and shallow breathing at all times. The ease of breathing preserves the patient's energy. The closed system preserves body fluids, as none are lost through the lungs; the reflex sweating of ether anesthesia is not seen. The gas that is breathed is warm and moist; the skin of the patient is always warm, dry, and pink. It is no longer necessary to change the gown because it has been drenched with perspiration during the anesthetic. It follows that body heat is preserved as none is lost by evaporation of excessive perspiration.

3. Depth of anesthesia can be regulated quickly because high concentrations of the gas can be inhaled without irritation to the larynx and without subsequent closure of the glottis and cords, which is nature's physiologic response to irritating vapors. This phenomenon makes endotracheal intubation easy and it can be accomplished in a shorter period of time than with ether.

4. The gas is non-toxic to the body tissues. The liver, kidneys, heart, and lungs present no evidence of damage after high concentrations of the gas have been administered.

5. The use of this gas for anesthesia allows a wide margin of safety. The lethal dose is more than twice the anesthetic dose, and a 50 per cent greater concentration is necessary to cause death after the stage of respiratory arrest has been reached.[19]

6 Cyclopropane has relaxing power approaching that of ether. Adequate relaxation for abdominal surgery is obtained in 85 per cent of cases. In those cases which fall short of good relaxation, the addition of small amounts of ether will usually provide the desired results.

7. Postoperative complications following abdominal operations are less after cyclopropane than the other commonly used anesthetic agents.[3,11,12] Distension is less after cyclopropane than ether because the tone of the intestinal tract is not lost and peristalsis continues. In this feature it possesses one of the advantages common to spinal analgesia. Respiratory complications are 50 per cent less than after ether.[3, 11, 12] The other less frequent complications are retention of urine, pyelitis, and parotitis.

8. Under conditions requiring special technic, with the aid of closed endotracheal cyclopropane anesthesia, the anesthetist can provide just about anything the surgeon desires. This is a great boon to thoracic surgery, usually done on the tuberculous patient. Because of decreased functioning lung tissue, the cramped position, and shallow breathing, the anesthetic mixture must contain a high percentage of oxygen and be non-irritating to the already diseased lung. Only with cyclopropane can this be accomplished. It is possible for the anesthetist to provide apnea, passive respiration, and positive pressure whenever called for during the course of anesthesia.

The results of thyroid surgery are much better with cyclopropane, as a high degree of oxygen can be maintained which is so necessary when the basal metabolic rate is high. In those cases with large glands producing some obstruction the high oxygen content and endotracheal catheter make for a better, smoother anesthetic.

Postoperatively, patients who have had cesarean sections do exceptionally well after cyclopropane anesthesia because there

is less nausea and vomiting, less blood loss due to better contractibility of the uterus and less distension because intestinal tone has not been lost.[16]

There are few contraindications to its use. Anemia, heart disease, hyperthyroidism, respiratory obstruction, and thoracic surgery present definite indications for cyclopropane. Most anesthetists have agreed that it is the anesthetic of choice to use on the so-called bad-risk patient. Remembering its limitations, and after weighing the evidence presented, it appears that cyclopropane approaches more nearly the ideal inhalation anesthetic than any other drug which is available at the present time.

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THE THROMBOCYTE IN ALLERGY

A PRELIMINARY REPORT*

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The presence of purpuric patches in five per cent of cases observed in the Department of Allergy at Charity Hospital suggested a possible relation of thrombocytopenia to allergy, especially so in patients with skin allergies with hemorrhagic spots. Many observers before had detected hemorrhages in different clinical entities associated with thrombopenia.

The presence of the platelet in the blood stream has been known fifty years but attracted notice only since 1922. Very little is known of the origin in the body of the platelet; the majority of the writers, however, accept the theory of Wright, who assigns this function to the bone marrow where particles are detached from the megalocytes. The platelet is supposed to liberate a substance having vasoconstrictor power, is formed in the bone marrow and is destroyed in the spleen. X-ray and radium raise the platelet count.

Wright and Minot have attributed to the platelets the chief source of thromboplastin, as a marked fall in the count attends practically all purpuras. Those interested in the function of the platelet will find valuable information from the pen of Allard, Piney, Osler, Frank, Rosenthal, Lippman, Zadek and Rudd. These observers were not interested in allergy especially, but an analysis of their work, as well as that of Shlack, definitely establishes the fact that recovery from disease is accompanied by thrombocytosis, a sign not only of recovery in acute cases but of definite favorable prognosis whenever observed. The object of my research was to ascertain whether or not recovery from an allergic spell is accompanied by a rise in the platelet count; the normal is 1 to 16 red blood cells.

Platelets however have such a short life (Duke) in the blood stream, and the high and low normal levels are so variable, that

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one hesitates to draw conclusions in a small group of cases (27 in all).

I was gratified to note that my cases as to course and prognosis, as shown in the schedules submitted below, confirmed the conclusion of former writers. I was curious to ascertain why the count showed such variation, what agents we could use to raise the count, and whether or not it was possible to prevent a recurrence of hay fever, asthma or skin allergy by inducing a thrombocytosis. The result of the counts only is submitted in this paper; I hope to report later on the balance of the observations.

CASE ANALYSIS

My cases are not imposing enough to establish definitely what low level in the count heralds an attack, or what rise points to recovery, but the figures given are sufficiently accurate and interesting to form a basis for future research and corroboration. I selected from the rich material offered by the clinic, at first 35 cases of the most serious forms of allergy but was able to gather complete data only in 27 cases distributed as follows:

TABLE I

Hay fever	5
Allergy	5
Asthma	16
Skin and allergic conjunctivitis	1

The first count was made during the spell, the second within two weeks of the spell. The result was as follows:

A. Hay Fever: All cases whether spring or fall showed an average rise of 55,600.

TABLE II

PLATELET COUNT IN HAY FEVER

Patient	Antigen	During Attack	In Convalescence
G. D.	Ragweed	325,000	350,000
G. P.	Ragweed (focus in teeth)	325,000	250,000
H. B.	Ragweed	250,000	375,000
M. P.	Ragweed	223,400	265,000
T. J.	Grass	212,940	273,780

B. Asthma: The spring variety showed an average rise of 21,086, the fall of 12,412. Both combined, an average of 17,231. The three cases which showed a fall in convalescence were: one case complicated with sinusitis, and two cases complicated with tuberculosis.

TABLE III

PLATELET COUNT IN ASTHMA

Patient	Antigen	During Attack	In Convalescence
B. H.	Grass (focus in teeth)	250,000	265,000
P. A.	Grass (focus in tonsil)	114,910	213,830
A. A.	Grass	195,000	235,000
A. S.	Ragweed	530,200	510,100
E. W.	Grass (tuberculosis)	389,640	237,500
V. M.	Ragweed	240,000	260,000
C. L.	Ragweed (rickets)	225,000	230,000
L. R.	Grass (aneurysm)	290,000	260,000
D. W.	Grass	350,000	250,000
M. N.	Ragweed (sinus)	306,400	295,000
F. R.	Ragweed	411,600	507,650
L. M.	Grass	205,000	270,000
N. J.	Ragweed	223,000	198,645
A. T.	Ragweed	400,000	360,000
I. E.	Grass (tuberculosis)	342,000	420,000 (vaccine given)

C. Skin allergy showed an average fall of 8,000. Eliminating one case where large doses of vaccine were used, the cases then showed an average rise of 27,000.

TABLE IV

PLATELET COUNT IN SKIN ALLERGY AND CONJUNCTIVITIS

Patient	Antigen	During Attack	In Convalescence
L. C.	Grass	250,000	300,000
O. D.	Ragweed	440,000	326,000
M. W.	Grass	225,000	375,000
R. G.	Grass	534,200	370,000
M. B.	Ragweed	250,000	275,000
R. S.	Conjunctivitis	210,000	230,000

There were exceptions to the above findings. These are shown in the following table:

TABLE V

2 cases of skin allergy, grass and ragweed.
1 case of asthma, ragweed with sinusitis.
2 cases of tuberculosis, with grass allergy.
1 case (ragweed) in a child.
1 case (grass) with an aneurysm.

Note *all* cases of hay fever (1 grass and five ragweed) without exception showed a rise during convalescence.

DISCUSSION

From an analysis of the cases submitted, I am convinced that improvement in uncomplicated asthma and hay fever is always indicated by a rise of the platelet.

One of my diabetic patients with hemorrhagic patches cleared up and stayed cleared up with liver extracts given hypodermically, due in our estimation to the increase in the platelets. The mysterious role which the spleen is supposed to play in hay fever and asthma may be accounted for by the influence of the spleen on the thrombocyte count as observed by Bedson, Bachman, and Hultgren; so also the study of Meyers, Maingot and Gordon on the spleen and those of Gordon, Alrutz, Norbell and Piette. Kercki and Peliczar have likewise made interesting studies of the platelet in anaphylaxis.

Squire and Madison found a direct relation between food allergy and thrombocytopenic purpura, the count falling with the postprandial rise of eosinophiles from certain foodstuff in a series of children. The reverse was noted on the withdrawal of the offending allergen. In view of the research of McLean and Caffey, however, the conclusions are not as certain as they would be in adults. Though the majority of the cases of Squire and Madison were between two and sixteen years of age, yet these conclusions deserve serious consideration and no doubt will be confirmed.

CONCLUSIONS

From the investigation so far made I present the following:

1. The allergic spell, whether the shock organ be the nose, bronchi or skin is, as a rule, indicated by a drop of the platelets in the peripheral circulation.

2. Improvement is usually indicated by a marked rise in the count.

COSTS OF PNEUMONIA.——The average case of pneumonia in New York City costs $167.60, according to "A Study of the Economics of Pneumonia" conducted by Hirsh. This figure, according to Hirsh, may be taken as a fair average for the costs of pneumonia care in large cities, especially in the northeastern states. If it is representative of the costs for diagnosis and treatment of pneumonia, the annual bill for this disease in the United States amounts to more than $75,000,000.

"Pneumonia accounts for well in excess of 450,000 cases of illness a year in this country," Hirsh said, "approximately 25 per cent of which are fatal. This toll exceeds that of any other communicable disease. It also contributes to many thousands of deaths annually which are officially attributed to other causes, and the extent of this contribution has never been accurately evaluated."

Both difficult and expensive to treat, pneumonia presents a serious medical problem. Because of its extensive duration (for many of the non-fatal cases) and complexity of treatment, it creates also a very definite economic problem. This study was made in an effort to determine the amount and nature of costs of caring for pneumonia. It was compiled from the records of 625 pneumonia cases, in five hospitals, one home nursing service, and home cases, in New York City. In each latter case, there were private physicians; of the hospital cases, 400 were ward, 66 semi-private, and 94 private.

Top costs ran up as high as $1,554, while as little as $5.50 was spent at the other extreme. Those cases treated in the hospital were generally much higher than those in the home, although the cost of ward care was almost as low as home care. The average cost for semi-private cases came to about $225; for private cases, $267, as compared with the low cost for the ward cases, $145.

Physicians' services were estimated at minimum rates, even though not actually paid for by patients in the ward. They constituted only 24 per cent of the total cost, with a somewhat higher percentage in semi-private and private cases.

The use of serum, which has reduced the mortality from pneumonia, has added substantially to costs. In the average case in which it was used, it amounted to $70.

These figures are especially significant in the light of recent findings of the National Health Survey which showed that more than half of the families in New York City have incomes below $125 a month. Thus, Hirsh pointed out, few persons can shoulder the costs of an unexpected illness which amounts to more than 10 per cent of their annual income.

Striking persons of every age, sex, color, and social group, pneumonia is the leading cause of death at the extremes of life. Forty per cent of the total number of deaths due to this disease are among men and women in the most economically productive period of life, those between 15 and 64.

"Their deaths," declared Hirsh, "rob families of breadwinners, industry of producers, and communities of consumers." Further, "there is a much lower death rate among professional and business men than among unskilled workers. Thus, the responsibility for the care of many of these cases falls upon the community since many individuals find treatment of the disease too expensive for them to hear."

Public Health Reports,
December 9, 1938.

A RESTRAINING BELT*

OSCAR W. BETHEA, M. D.†
NEW ORLEANS

Many devices have been employed for keeping the unwilling patient in bed. Most of these entail a measure of restraint that is uncomfortable, and may be very much resented by the victim in his more lucid moments.

The restraining belt here presented has been used for several years at the Baptist and Charity hospitals in New Orleans and the following advantages have been demonstrated: First, it is effective. No patient has yet freed himself from this restraint. Second, it allows free movement of the arms and of the lower extremities and some use of the body. Third, it is free from the possibilities of injury especially when applied over the sleeping garment. Only a few minor cases of chafing have been noted. Fourth, it has occasioned a minimum of resentment on the part of the patient.

This device consists of a band that is to be drawn tightly across the mattress and under the chest of the patient and fastened on each side of the bed by a lock buckle. Attached to this are two pieces that meet in front of the chest and fasten with a lock buckle. A shoulder strap on each side prevents the patient from working himself up and out. The belts that have been used experimentally were rather crudely made in a local shop. Having proved an effective yet kindly appliance, they are being put on the market (unpatented) by A. S. Aloe Company of St. Louis, Mo.

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*Presented at a meeting of the Orleans Parish Medical Society, November 14, 1938.

†From the Department of Medicine, Tulane University.

Fig. 1. Restraining belt properly adjusted.

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THE NEW YEAR

The new year dawns, apparently with a few bright rays of light shining through the dull morning clouds. Business on the whole seems to be on the up-grade. People are buying more than they did, and reports from all parts of the state indicate that they are paying their doctors' bills a little more cheerfully, and happily and promptly than at any time in the last nine years except for a brief few months about a year ago. Perhaps the period of depression in the United States is passing; perhaps we are in a chronic state of depression from which we, and all other peoples of the world who are in a similar fix, may never emerge. In spite of the depression, however, it is safe to gamble that the medical profession of the State of Louisiana will enjoy life, and that their life is well worth while, not only to themselves and to their families, but also to innumerable people to whom they give service. After all, life is what you make of it yourself, and in spite of financial worries, family griefs or other troubles, to paraphrase an old saying, "A man worth while is a man who can smile," and doctors can and do smile despite night calls, harrassing patients and punctured tires.

May the New Year be the best year you have ever enjoyed, is the earnest wish of the New Orleans Medical and Surgical Journal and of those connected with it!

PERSECUTION AND PROSECUTION

The American doctor leads a more or less isolated life. He comes in contact with many people, it is true, but they are usually the sick who are primarily interested in their illness, and most of them are women. He probably reads in the paper that men in business, particularly "big business," have been complaining bitterly about the interference of the Government in their affairs. He does not realize, however, that Governmental interference may possibly be a tremendous factor in reducing the efficiency of the business man, besides subjecting him to a tremendous amount of expense in order to make reports, to get up figures and to fill out forms. Raymond Moley, in an editorial in News Week a short time ago, said it is a great pity that "a man like Wilke, president of the Commonwealth and Southern, and other business men, has been compelled to turn away from the process of expanding business enterprise and to re-employ millions of the unemployed . . . that he has been compelled to spend his great talents in an unproductive controversy."

Now it is the turn of the medical profession to be assailed. It is about to be investi-

gated through its chief medical organization. The men who are officers in this organization have been compelled to stop their usual productive activities in order to prepare for a subpoena relative to the District of Columbia case. In a recent visit to the headquarters of the Association it was found that the men at the head of several important divisions were spending day and night, to the detriment of their regular duties, in order to collect a tremendous amount of data and facts relative to the relations of the American medical profession to the individual medical man. Not only were all files of many years back subpoenaed but even such minutiae as intra-departmental communications subpoenaed. The time and energy required to get these filed papers together are terrifically demanding. All this simply because the Federal Government has decided, through a few cockbrained individuals, that the American Medical Association controls in every way, form and manner the practice of medicine in the United States. This subpoenaing of records is probably entirely a fishing expedition. It is hoped that something will be found against the American Medical Association which will substantiate what these few Washington officials think is true but about which they have no proof whatsoever, nor will any proof be brought forth about something which is not true.

Nor is this all. Recently over one of the big radio chains a debate was held between the editor of the Journal of the American Medical Association and a representative of the Department of Justice at Washington. In this debate the Washington speaker tried and convicted the American Medical Association before a trial had taken place. This certainly is a remarkable and deplorable state of affairs, that the Government through its trial officials should attempt to prejudice a case even before this case had appeared before a jury is almost unbelievable and unthinkable.

The above paragraphs may seem like a tirade against the Federal Government or rather a few of its employees. It is, but it is well deserved. A few Federal Government officials are apparently firmly convinced that state medicine should be introduced at a very early time. In order to have the backing of the American people, which they undoubtedly do not have at the present time, they are attempting by propaganda and by half truths to confuse the people by contending that the way American medicine is conducted at the present time is harmful to the individual and to the population as a whole. When, as and if, through their propagandizing channels they may be able to give the American public a distorted idea of medical practice, then they feel the time will be ripe to spring state medicine on the American voter.

This is really most lamentable. American medicine needs no defense. One of the important officials of Washington has just pointed out that the death rate last year was lower than it had been in this country for many years. The health of the population as a whole is guarded most effectively by present day methods. The people of America receive better medical care than any country in the world. There is never a serious illness or epidemic which is not taken care of adequately. If the American man, woman or child is not getting medical care it is either because of their unwillingness to make use of the existing facilities or else it is sure to be some minor condition which is of minimum moment. Did any one in Washington ever hear of an indigent patient with a broken leg or a pneumonia or an acute appendicitis who does not receive the same care, attention and skill that a person would receive who can afford to pay?

SMOKING

The scientific physician can prove that tobacco smoking may have unfavorable effects on the human being. The smoker feels that these effects are minimal and that they are overshadowed by the pleasure derived from inhaling a cigar, cigarette or a pipe. Of course, ultimately this smoking becomes a habit, broken sometimes with difficulty, and rarely.

One of the untoward effects of cigarette smoking which has not been generally

recognized is described by Segal[1] in a recent article. He recounts briefly the history of six patients who complained of chronic fatigue and ease of tire. None of these people could be called heavy smokers except, perhaps, one. When they stopped smoking, in a period of a very few weeks, fatigue disappeared, energy increased and they gained weight.

Nicotine is the substance in the smoke that produces the systemic effect. The local effect, such as morning cough and irritation of the throat, is produced by pyridine and its compounds. Nicotine is a vasoconstrictor that raises the blood pressure, increases pulse rate and decreases skin temperature. It is possible that the fatigue may depend upon the primary rise of pulse rate and increase in blood pressure, which is succeeded by a fall in pressure. Noticeable effects are not perceived by any means in all people. They are very much more likely to take place in those who are under fifty years of age than in the older generations. This statement is substantiated by a series of studies on young and old people after a cigarette. In the young people, the increase in pulse rate was as high as 50 per minute, and the average in the group was 21. In the older individuals, 7 of the 11 showed no increase in heart rate, and the average increase was only four beats per minute as contrasted with the group under thirty-five, in which it was 23. The electrocardiographic studies showed that there was a decrease in the height of the T-wave in the young group, which was very slight in the older group. Incidentally, Segal notices that there was no difference in the effect of the standard brands of cigarettes as contrasted with two denicotinized brands. He studied, also, electrocardiograms to determine the efficiency of the so-called filters, and found that there are the same effects produced as with the unfiltered smoke. He concedes, however, that where the filter may remove some of the nicotine it does not remove enough, because, while 3.33 mg. of nicotine are taken in by the body when a cigarette is smoked, even the most effective filter cannot eliminate the 1 mg. of this drug which can produce a systemic effect. The assumption on which Segal explains fatigue that occurs in some individuals after smoking is that smoking produces a rise in blood sugar; this in turn depletes the muscle glycogen, a loss of potential muscular energy. Excessive or repeated cigarette smoking continues to remove muscle glycogen, leaving the muscle tired and exhausted. The change in T-wave "represents a reaction of the heart to increased work," which, the author believes, does no good and probably does harm over a period of time.

It would be interesting to have had a more detailed analysis of the effect of smoke on the two sexes. Women are thought to be more frequently affected than men by the action of nicotine or by small doses of the substance. There is a certain amount of experimental evidence to prove this, but it is not brought out in this paper.

Irrespective of whether or not nicotine does do harm, possibly the evil effects of smoking are counteracted by the rise in blood sugar, by the soothing of the nerves and by the enjoyment of smoking. The physician cannot say ex cathedra that smoking harms everyone; he should judge the individual patient and whether it is advisable for that patient to smoke or not to smoke.

1. Segal, H. L.: Cigarette smoking: 1. As a cause of fatigue. 2. Effects on the electrocardiogram with and without the use of filters, Am. J. Med. Sci., 196: 851, 1938.

HOSPITAL STAFF TRANSACTIONS AND CLINICAL MEETINGS

HIGHLAND SANITARIUM

Shreveport

The Staff of the Highland Sanitarium held its monthly meeting at 8 p. m., December 15, 1938, in the Clinic Building, following dinner in the Sanitarium dining room.

The following officers were elected for the coming year: Drs. T. B. Tooke, president; M. M. Collins, vice-president; H. W. Boggs, secretary.

SCIENTIFIC PROGRAM

Dr. Dean H. Duncan gave a very instructive talk regarding the newer treatment of mental disorders, mentioning some of the history in regard to the discovery of hypoglycemic shock with insulin. Dr. Duncan then discussed the method of giving insulin therapy and metrazol therapy, including the mechanism accounting for the good results, the results up to the present time being 28 per cent in old cases and 90 per cent in early cases. The bad results were also mentioned and the treatment of complications.

Dr. J. A. Hendrick presented a most interesting report of the recent meeting of the Eastern Branch of the Urology Association of America. Dr. Hendrick discussed several papers on such subjects as the treatment of recurrent kidney stone, the dissolution of stones, interstitial cystitis of the bladder, tumors, and a paper on surgery of the prostate by Dr. Young.

PAUL W. WINDER, M. D., Sec.

SHREVEPORT CHARITY HOSPITAL

Shreveport

The meeeting of the staff was opened at 6:40 p. m. on November 15, 1938, by the President of the Staff, Dr. J. M. Bodenheimer.

The minutes of the previous meeting were read and approved as read.

SCIENTIFIC PROGRAM

1. A case of congenital displacement of the kidney with marked nervous symptoms. Dr. Johnston narrated the history and course of the case and Dr. Gill presented a discussion of the case.

The nervous symptoms, which began in childhood, became progressively worse until on last admission the patient was entered in hysterical coma. Hysteria, crying spells, and pain in lower left quadrant predominated. Pyelogram showed the left kidney to be over the promontory of the sacrum. P. S. P. and other tests showed normal function of the ectopic kidney. Relief was not obtained by oral administration of urinary antiseptics, and cystoscopic drainage gave only temporary relief. Since the grave nervous symptoms were due to the ectopic kidney, operation was decided upon. Following operation by anterior route with removal of the kidney, nephropexy being impossible, the nervous and mental conditions were relieved immediately and patient's outlook has been splendid since then.

Discussion by Drs. Potts, Mays and Winder.

2. The second case, an unusual clinical manifestation in amebic abscess of liver, was presented by Dr. Campbell.

No history of diarrhea or jaundice obtainable. No fever present throughout course of treatment. Large mass over liver, fluctuant anteriorly. Aspiration was done and about 4,250 c.c. of dark greenish fluid was withdrawn, and amebic cysts were demonstrated in the fluid. Emetine hydrochloride 1-1000 was used to irrigate the abscess cavity. The patient was given emetine hydrochloride, grains 1, daily for 7 days. Response to therapy was good.

Dr. Potts discussed a similar case which was treated by surgical drainage, plus emetine hydrochloride grains 1 daily. Discussion also by Dr. Cassity and Dr. Weber.

3. The third case was for diagnosis, and was presented by Dr. Jones. Impression on admission was pneumonitis and streptococcic sore throat; later suggestions were bacterial endocarditis and rheumatic fever. Felix-Weil reaction negative for typhus fever; malaria smears negative; no response to quinine sulfate; agglutination negative for typhoid and brucellosis. Fever responded to quinine dihydrochloride and quinidine. Murmur is still present over apex of heart; joint swellings have subsided and spleen no longer palpable.

Discussion by Drs. Boyd, Mays, Shavin and Eaddy. Tularemia, undulant fever and septicemia believed not entirely ruled out.

Dr. Bodenheimer urged the various committeemen to be active in the interest of improving hospital service.

W. M. HALL, M. D., Sec.

HOTEL DIEU

New Orleans

The regular monthly meeting of the Staff of Hotel Dieu was held in the Nurses' Lecture Room of Hotel Dieu on Tuesday, November 22, 1938.

The meeting was called to order by the President, Dr. A. D. Mouledous, and with the Secretary, Dr. C. E. Gorman, at the desk.

Dr. John Couret presented a fatal case of estivo-autumnal malaria with autopsy findings, gross and microscopic.

Dr. C. J. Tripoli: This case was selected for presentation because of the rarity of occurrence of this particular disease in an obese individual. None of the usual causes of heart enlargement are obvious and after considering all etiologic factors we find that this patient fulfills the criteria for diagnosis of a chronic deficiency (beri-beri, B_1), heart disease with left and right ventricular dilatation, probable right auricular dilatation, occasional ventricular ectopic rhythm; type II B.

The anemia in this case was obviously produced by sulfanilamide. Further proving the accepted diagnostic criteria as correct was the complete alleviation of symptoms on specific therapy consisting of iron, vitamin B_1 and liver extract.

In discussing this case, the frequency of this disease in our every day practice and the dramatic disappearance of symptoms under specific therapy were stressed.

A review of the physiologic mechanisms and the pathologic lesions characteristic of this disease were also presented.

CASE REPORT

The patient complained of shortness of breath and swelling of the feet and legs.

Since January 1938 this patient has had edema of the feet, ankles, legs and thighs present all the time but diminished slightly on arising in the morning. There was also dyspnea on slight exertion and fullness in the epigastrium following meals. The edema has become progressively worse despite rest in bed and digitalis. At no time has there been any complete alleviation of the patient's symptoms during his illness.

Complete review of systems reveals no noteworthy factors except those usually accompanying persistent congestive heart failure.

In November 1937 a prostatic resection was undertaken for urinary infection and prostatic hypertrophy. Following operation the infection was controlled by sulfanilamide which was necessarily given over a period of one month. During this time diet consisted of broth, fruit juice, sweet drinks, jello, and potatoes, due to lack of appetite. The past history presented nothing of note except a dysuria for the previous several years for which operation was undertaken.

No factors of diagnostic importance were noted in the family, social or marital histories.

Physical examination revealed a well developed and nourished white male, 65 years of age, lying in bed on a back rest, not acutely ill. The temperature was 98.6°F., respiration 20, pulse 80, B. P. 100/70; weight 210 pounds, height five feet, seven inches. There were no abnormalities noted in eyes, ears, nose and throat. Neck revealed engorged and pulsating external jugular veins. The veins of the chest were visibly distended despite the patient's obesity. Expansion was bilateral, equal and symmetrical. The lungs showed dulness on percussion and crepitant rales present at both bases. Apex beat not visible. On palpation apex beat could be elicited 15 cm. to the left of the midline in the fifth interspace. Heart sounds were distant and a low, barely audible systolic murmur could be heard over the apex of the heart. This murmur was not transmitted. There was no demonstrable evidence of arteriosclerosis of the peripheral vascular system, including the retinal vessels. The abdomen was protuberant and free fluid demonstrable in both flanks. Liver, spleen, and kidneys were not palpable. The genitalia showed no external clinical abnormalities. The reflexes were present, equal and symmetrical, but markedly diminished. The extremities showed a bilateral tenderness over both calf muscles.

Laboratory: Blood: red blood cells 3,100,000; hemoglobin 60 per cent (Sahli); white blood cells 6,000; differential, normal. Red blood cell changes were consistent with normocytic hypochromic anemia. Blood chemistry: no abnormalities. Blood Wassermann: Negative. Total blood protein: 6.2 gm./100 c.c. Urinalyses showed no abnormalities except trace of albumin and few pus cells per low power field. P. S. P. intramuscularly 28 per cent and 23 per cent respectively on two different occasions. Stool examination: No occult blood, no larvae, ova or cysts of parasites. X-ray examination: Genito-urinary tract; no evidence of urinary calculi. Chest: Heart markedly enlarged to the left; lungs and aorta normal; heart measurements; transverse diameter of the chest 30 cm; transverse diameter of the heart 18.8 cm. Electrocardiogram: Q_1, Q_2 and Q_3 present; slurring in all leads, notching at the apex in lead I; occasional left ventricular extrasystoles; T waves low in all leads.

Diagnosis: Chronic deficiency (beri-beri or B_1); heart disease with left and right ventricular dilatation; probable right auricular dilatation, and mitral incompetency, occasional ventricular ectopic beat, type II B.

Progress: After ten days of specific therapy edema disappeared. Patient lost eleven pounds in weight and at the end of one month cardiac transverse measurement was 16 cm. in diameter and blood pressure 120/90. Red blood cell count was 4,500,000. Patient up and about and performing routine duties.

J. T. NIX CLINIC

New Orleans

At the meeting held in October, Doctor J. M. Perret presented the following paper:

PRIMARY CARCINOMA OF THE LIVER
REPORT OF THREE CASES

(Continued from November Issue)

Case 1. M. C. E., a white male of 49 years, a rice buyer by occupation, was admitted to Hotel Dieu, New Orleans, on December 7, 1929, complaining of vomiting. The present illness began on November 16, 1929, when he vomited everything that he ate. The vomiting stopped when he took soft diet, but recurred again after a week. He has not had blood in the vomitus or in the stools. There have been no abdominal pains. The appetite was fair and the bowels were regular.

Past History: He has had the usual childhood diseases. Influenza in 1918 but no other serious illness. He has never been operated on.

Physical Examination: Nutrition was fair. The scalp, eyes, ears, thyroid were negative. The mouth was in good condition. Chest: The heart and lungs were negative. Blood pressure 100/60. Abdomen: There was a mass in the epigastrium about the size of a hen egg. The liver was somewhat tender. The spleen was not felt.

Laboratory Examinations: Urine: There was a slight trace of albumin; hyaline, finely and coarsely granular casts and a few pus cells were

found. Phenolsulphonephthalein test: first hour, 35 per cent; second hour, 20 per cent. Blood: total white count, 21,800; differential count: small lymphocytes, 14; large lymphocytes, 2; neutrophiles, 84. Malaria, negative. Blood Chemistry: Non-protein nitrogen, 60 mg. per 100 c.c.; urea nitrogen, 30 mg. per 100 c.c.; creatinine, 2.7 mg. per 100 c.c.; sugar, 96 mg. per 100 c.c.

X-ray Examination: Chest: heart, lungs and aorta were normal. Esophagus was negative. Stomach: normal in outline and motility, high in abdomen, fair size six hour residue, moderate twenty-four hour residue. Duodenum: first portion, dilated, normally filled; second portion, markedly enlarged with evidence of incomplete obstruction at the junction of the second and third portions. Cecum, slightly tender, not freely movable. Appendix, not seen. Colon, negative. Conclusion: Incomplete obstruction of duodenum and appendicitis.

Operation was performed by Dr. J. T. Nix, December 14, 1929. Properative clinical diagnosis: probable carcinoma of the stomach. X-ray diagnosis: incomplete obstruction of duodenum; appendicitis.

A large incision was made in the mid rectus to right of median line above umbilicus, beginning from a point at level of umbilicus and extending to costal border. On opening the abdominal cavity the stomach and the duodenum presented one large tube. The pylorus was about two and one-half inches in diameter. A hard mass was felt on the surface of the liver. A posterior gastroenterostomy was done with linen sutures. The patient began to do badly when the operation was about one-half finished. In spite of all efforts he succumbed as the final sutures were being placed. Before his death the liver was visualized and found to be completely filled with large areas resembling carcinomatous growths; one about four inches in diameter and the size of a closed fist was present on the under surface of the liver.

Autopsy report on body of M. C. E. by Dr. M. Couret: Abdominal cavity: the omentum and peritoneum showed nothing of special note. Spleen was examined in situ and was slightly larger than normal and moderately filled with concretions. Kidneys were examined in situ and appeared normal in size. Liver was found to be slightly larger than normal but studded throughout with large tumor masses. These varied in size from that of a pin head to that of a small orange, very firm in consistency and pearl gray in color with yellowish gritty centers in the larger tumors (necrosis).

Anatomic Diagnosis: Carcinoma of the liver (primary).

Microscopic Examination: Microscopically, the liver showed extensive infiltration of a neoplasm, showing an atypical picture of glandular acini and ducts. The character of the epithelium lining these ducts was very irregular with large, small, and multilobar nuclei. The cancerous cells belong to Brodie's III and IV types. Mitosis was extensive and there was much necrosis present. The fibrous tissue reaction to the neoplasm was scant, so that it is difficult to outline where the tumor ended since infiltration of the cancer cells through the remaining liver tissue was extensive. The remaining liver tissue showed cloudy swelling, moderate fatty degeneration and marked pressure atrophy of the cells nearest to the invading neoplasm. There were patches of round cell infiltration (lymph and plasma cells) scattered throughout the liver tissue and less so in the stroma supporting the cancer cell. The portal tracts also showed moderate round cell infiltration and fibrosis. There was no definite cirrhosis found. The neoplasm would appear to have started in the bile radicals or the smaller ducts.

Diagnosis: Primary adenocarcinoma of the liver.

Case 2. DeJ. H. J., a white male of 72 years, engineer and machinist, was treated in 1925 for a bronchitis which had been chronic with him since childhood. In 1921 he had had a dry pleurisy which had incapacitated him for a week. As a young man he had had urethritis. Habits: He drank one-half gallon of beer a day (worked in a brewery) and smoked twelve cigars. Up to four months previous he had smoked twenty to thirty cigarettes a day.

Family History: One brother died of tuberculosis.

Physical Examination: The heart showed an accentuation of the aortic second sound. There was peripheral arteriosclerosis. Blood pressure, 162/86. Lungs showed distant breath sounds over the back but no rales. Abdomen was prominent, puffed out and probably had some ascites. The viscera were not palpable.

Laboratory Examinations: Urinalysis, negative. Phenolsulphonephthalein test: 200 c.c., 50 per cent in two hours. Blood: total white count, 7,000; differential count: small lymphocytes, 30; large lymphocytes, 2; neutrophiles, 68. Wassermann, negative. Sputum, negative for acid fast bacilli.

X-ray of Chest: Heart and aorta, negative. Lungs, irregular increase of density extending well to periphery of lungs. Increased density of right apex. Some linear markings in the left apex. Areas of calcification in hilus region. Conclusion: Evidence of tuberculosis.

X-ray of Gastro-intestinal Tract: Evidence of chronic appendicitis.

On April 28, 1930, the patient had an appendectomy with an uneventful recovery.

From October, 1930, until September, 1933, the patient was not seen. On September 27, 1933, we saw the patient at his home as he was again suffering from bronchial symptoms and had had fever for several days. During the succeeding week he had several attacks of hemoptysis, amounting to a few ounces of blood. The patient's general condition at this time was very poor. He was

extremely emaciated and had lost about twenty-five pounds during the previous year.

Physical examination revealed the physical signs of chronic bronchitis with bronchiectasis and consolidation of the left lung. The heart showed nothing of note. The blood pressure was 140/100. Abdomen: The liver was enormously enlarged, extending as low as the iliac crest. The organ was hard and nodular.

Clinically there was no doubt that we were dealing with a malignant liver but we failed to realize that it was primary until this was revealed to us at autopsy.

The patient was admitted to Hotel Dieu on November 2, 1933, and died two days later. The symptoms and signs of bronchopneumonia and hepatic toxemia closed the clinical picture.

Autopsy by Dr. M. Couret: The body was that of a greatly emaciated, but normally developed white male, body length approximately 170 cm. Postmortem rigidity was only moderately marked. Lividity was present on the dependent parts. Pupils were equal, measuring 2 mm.

The abdominal cavity: on opening the abdomen about 300 c.c. of bloody turbid fluid with a few small blood clots was found. The peritoneum and the serous coat of the intestines were smooth and glistening. The omentum was curled over the stomach and transverse colon but was not adherent to any structure. There were no adhesions between the intestines and any other abdominal organs. The mesentery lymph nodes were not enlarged, though there were several enlarged nodes, retroperitoneal and especially in the region of the pancreas and liver. The appendix was retrocecal and resembled a thick cotton cord.

The thoracic cavity: both pleural cavities contained approximately 100 c.c. of slightly blood-tinged fluid. There were old adhesions of the apices of both lungs and a slight roughening of the pleura at the base of the right lung. The right lung showed marked anthracosis and a mottling with dark bluish red areas here and there throughout the lower lobe. On section the organ showed dark red and reddish gray areas that were markedly consolidated at the base of the organ. On pressure a large amount of red, frothy fluid came from the tissues not consolidated. The left lung had the same general appearance as the right excepting that there were no consolidated areas and a greater amount of red, frothy fluid came out on pressure. Both lungs showed old scars and calcified tubercles at their apices.

The pericardial cavity: this contained approximately 50 c.c. of clear straw colored fluid. There were no adhesions to the pericardium. The heart showed a slight milkiness of the epicardium and extensive tortuosity of the epicardial vessels. The heart muscle was of a dead leaf color and somewhat friable. The cavities of the organ were not examined, excepting the pulmonary artery which was opened in situ and only postmortem clots found. The heart is slightly larger than the average.

The liver was markedly enlarged, the border of the right lobe extending on a level with the crest of the ilium. The organ was of a light brown color and there was scattered throughout a large number of neoplasms, varying in size from a pea to a hen egg. The larger areas were markedly necrotic. The normal markings of the liver were present, the organ having a nutmeg appearance. The entire surface of the liver showed small petechial hemorrhages from which there was evidence that bleeding had occurred producing the hemorrhage found in the abdominal cavity.

The gallbladder was almost entirely obliterated by a mass of small stones, gritty material and inspissated bile. The wall was markedly thickened and the duct appeared almost completely occluded. The bladder was at its largest point not larger than a pencil. The common bile duct, however, was patulous. The spleen was of normal size, the capsule was gray and somewhat wrinkled. On section the pulp was of a light wine color and the normal markings indistinct.

The gastrointestinal canal: the stomach and intestines were carefully examined along their entire length and showed nothing of note, excepting petechial hemorrhages throughout the mucosa of the stomach which contained a moderate amount of "black vomit."

The pancreas was negative, though about it were several lymph nodes that were markedly enlarged and varied in appearance, and on section were firm in consistency.

The kidneys were of average size and, though each showed marked congestion of all vessels, no definite gross pathologic changes were found. The adrenals were negative and showed only moderate postmortem autolysis. The ureters were negative. The bladder was half filled with urine and showed nothing of note.

The vascular system was not disturbed.

The brain and spinal cord were not removed.

Anatomic Diagnoses: Carcinoma of the liver (primary); metastasis in adjacent lymph nodes; infarcts of the lung; capillary hemorrhage; cholelithiasis; chronic apical bilateral tuberculosis.

Microscopic Examination: The tumor of the liver with metastasis showed a carcinoma with alveoli containing round and moderately hexagonal cells. The cells varied in size and some contained larger and smaller nuclei. There was marked mitosis present. The type of cell suggested more probably the origin of the neoplasm. The surrounding liver cells showed different stages of degeneration and atrophy and there was some scattered thickening of the intima of some of the vessels especially of the portal tract. The ducts and gallbladder showed no evidences of tumor formation. There was considerable necrosis present in the larger tumor masses.

Diagnosis: Primary carcinoma of the liver (origin probably bile radicals); metastases in adjacent and surrounding lymph nodes.

Case 3. B. I., a white male of 70 years, Swede, a religious by profession, was admitted to Hotel Dieu on September 3, 1938, complaining of shortness of breath and general weakness. The present illness dated back three weeks. He had had a mild cough, fever, nausea and general weakness. Previous to present illness he claimed good health.

Past History: He had had measles, mumps, pertussis and pneumonia. He had never been operated on.

Family History: No tuberculosis, cancer, renal disease or diabetes.

Physical Examination: The patient was a well developed and nourished white male. Mentality was normal. He was dyspneic. The skin was icteroid. The glands and reflexes were normal. There were large varicose veins of the left leg. Pupils were equal and reacted to light and accommodation. Mouth: There were few teeth left and caries were present. Pyorrhea alveolaris was noted. The tongue and tonsils were negative. Chest: There was flatness over back of the chest from the level of the angle of the scapular downward. Both tactile and respiratory sounds were slightly diminished over this area. Heart was slightly enlarged and showed gallop rhythm. Peripheral arteries showed arteriosclerotic changes. Blood pressure, 130/80. Abdomen, no tenderness, rigidity or ascites. The liver was enormously enlarged, hard and the edge was irregular and extended more than a hand's breadth below the costal arch. The other organs were not palpable. The genitals were negative.

Laboratory Examinations: Urine—faint trace of albumin; many granular casts and many pus cells. Phenolsulphonephthalein test: 35 per cent first hour; 20 per cent second hour. Blood—total white count, 26,000; differential count: small lymphocytes, 11; neutrophiles, 89. Total red count: 3,590,000. Hemoglobin, 70 per cent Wassermann test negative.

X-ray Examinations: Chest: lungs were normal; heart and aorta were normal; considerable elevation of the right diaphragm suggesting subdiaphragmatic abscess. Gastrointestinal tract; esophagus, negative; stomach showed pressure to left from the mid line; irregular filling defect of the greater curvature of the pars media; duodenum filled and emptied normally; cecum, not tender, movable; appendix was not seen; colon was irregularly filled. Large hard tumor mass was palpated in the upper abdomen not connected with the stomach. Conclusion: Evidence of tumor of the liver and possibly of the greater curvature of the stomach (pars media).

Electrocardiogram: Q and Q_2 present; long P-R interval for heart rate of 100; diphasic P_3; inverted QRS in Lead III with notching towards apex; slurring R_1, R_2 and R_3; T-P interval short at times; nodal rhythm at times; P-R, 0.2 sec.; QRS, 0.08 sec. Rate: Auricular, 100; ventricular, 100. Rhythm, irregular at times. Definite evidence of myocardial disease.

Clinical Course: Patient's temperature reached 101.3° F. once. Most of the time the temperature was normal. The patient steadily and rapidly progressed to a fatal termination. The clinical course was characterized by weakness and increasing jaundice. He vomited several times in the last few days of his illness and the vomitus contained apparently nothing but normal gastric contents. He died eleven days after his admission to the hospital. There was no doubt in the minds of the attending surgeon and internist that they were dealing with a case of primary carcinoma of the liver. There was no history pointing to any malignancy of the stomach or amebic dysentery.

The treatment was symptomatic. The poor general condition of the patient, his advanced age, as well as the extensive pathologic involvement of the liver did not justify any surgical procedure. It was also felt that little good would result from deep x-ray therapy and so this was not used. It is regretted that an autopsy could not be secured.

SUMMARY AND CONCLUSION

1. A brief review of the literature has been made to show the incidence of primary carcinoma of the liver.
2. The etiology, pathology and clinical course of the disease have been discussed.
3. All our cases were in males.
4. The patients were old, 49, 70, 72 years of age.
5. All presented themselves for treatment, when the disease had progressed so far that cure was impossible, even had a correct diagnosis been made when they applied for treatment.
6. The diagnosis was made on the operating table in Case 1, at autopsy in Case 2, and at the bedside in Case 3.
7. A plea is made to aim at early diagnosis, because this is the only way that radical surgical treatment can be instituted with the hope that a cure may be obtained in some of the cases.

HUTCHINSON MEMORIAL CLINIC

New Orleans

Scientific Session conducted by the Department of Medicine, Dr. John H. Musser, presiding.

Peptic Ulcer in a Girl Fourteen Years of Age (Dr. Norton W. Voorhies): This little girl was first seen in the Clinic on November 10, 1938. She has been having localized epigastric pain for two years. The pain is sharp and sticking in character and occurs from one and one-half to two hours after meals. It is associated with nausea, belching and the passing of gas per rectum. The pain is relieved by the ingestion of food or sodium bicarbonate. During the past two months the pain

has occurred daily and has frequently awakened her from sleep at night. There has been no vomiting. She has not observed blood in her stools. There has been a five pound loss in weight.

On review of the past medical history we learn that she had whooping cough at nine months, measles at three years and mumps at eleven years of age. Her tonsils were removed at the age of seven.

The patient's father and mother, aged 53 and 43, respectively, are living and well. Her three sisters are in good health. She has four brothers, one of whom had a perforated gastric ulcer at 17. There is no history of diabetes, tuberculosis or heart disease in the family.

On physical examination we find an intelligent white female of about 14 years of age. She is well nourished and of the asthenic habitus. The blood pressure is 115/75 and the pulse 84 per minute. Nothing abnormal is found on examination of the head and neck. The thorax is long and somewhat flattened antero-posteriorly. The heart is of normal size; no murmurs are heard. The lungs are clear on auscultation. The abdomen is scaphoid in type and tenderness is demonstrable on deep pressure in the epigastrum. Nothing abnormal is found on inspection of the external genitalia. The extremities are well developed.

The usual laboratory work was done. The red blood cell count is 4,230,000 with a hemoglobin of 80 per cent. The white blood cells number 10,240, with a differential count of 76 per cent neutrophiles, 21 per cent lymphocytes, and three per cent monocytes. The urinalysis and the Wassermann reaction are negative. Gastric analysis after an Ewald test meal showed a combined acidity of 78 and a free hydrochloric acid of 58 and a positive reaction for occult blood.

The patient was sent to radiology for a complete gastrointestinal study. Fluoroscopic as well as x-ray examination showed a deformity of the duodenal cap. The roentgenologist recommended that she return after a course of belladonna in order to rule out a functional spasm of the duodenum. She was given tincture of belladonna Mx three times a day for five days and then the examination was repeated. The second fluoroscopic and x-ray study showed the same duodenal deformity and also a six-hour retention of barium in the stomach.

This case is of unusual interest for two reasons: first, because it is one of chronic duodenal ulcer in a girl of 14 years of age and second because there is a history of ulcer in the immediate family.

I reviewed the literature and found that there were 31 cases of chronic duodenal ulcer and six of chronic gastric ulcer reported in children (14 years of age and below) from this country. The course of the illness is quite similar to that of the adult. The pathology is similar and as extensive. Children are as likely to have complications. Bleeding and pyloric obstruction occur and perforation is not uncommon. Fosheé reports 13 instances of perforation in 17 cases of gastric ulcer studied. The child responds well to medical and surgical treatment.

It is interesting to note that this child's brother had a perforated gastric ulcer. One is impressed on reading the literature with the high familial incidence of this disease. Riecker, after a study of 942 cases of duodenal ulcer, found a family history of ulcer in 13 per cent.

Situs Inversus Viscerum — Case Report (Dr. George E. Burch): F. F., a 22 year old negro male, was admitted to the Hutchinson Memorial Clinic on September 8, 1938, complaining of dyspnea and palpitation. The dyspnea was of the sighing type and was not related to exercise. It was most frequently associated with nervous tension and financial or marital difficulties. During these periods he would also suffer from palpitation, consisting of tachycardia or a "skipping" of a heart beat. He suffered from dyspepsia from time to time but as a whole the rest of the history was essentially irrelevant.

On physical examination it was found that the patient was normal in all respects except that he had the findings of situs inversus viscerum, the heart being on the right side, the hepatic dulness on the left and the stomach tympany on the right. Laboratory studies were made and found to be normal except the teleoroentgenogram of the heart, the gastrointestinal series and the electrocardiogram. The teleoroentgenogram showed the heart to be on the right side but otherwise normal and the gastrointestinal series showed the stomach and intestines to be reversed in position as found in situs inversus viscerum. The electrocardiogram presented the characteristic changes of dextrocardia.

The electrocardiogram in congenital dextrocardia of situs inversus viscerum is so characteristic that the diagnosis of dextrocardia can be ventured on that finding alone. In Lead I all complexes, P, QRS and T, are inverted. Lead II has the characteristics of the ordinary Lead III, while Lead III has the characteristics of the ordinary Lead II. The reading of the electrocardiogram is facilitated by reversing the normal electrode, viz., by placing the right arm electrode on the left arm and the left arm electrode on the right arm and then applying to the resulting tracings the rules and interpretations as exist for the regular electrocardiogram. Occasionally, one may erroneously suspect a dextrocardia from the electrocardiogram, when, because of a technical error, the arm electrodes are reversed when applied to a subject with a normal heart. Such errors should be ruled out before venturing a diagnosis of dextrocardia on the electrocardiographic findings.

Hypertension with Pigmentation (Dr. Julius Lane Wilson): This woman, a Jewess, is 42 years of age, single, and was admitted to the Clinic on November 28, 1938, complaining of pain in the

right lower part of the abdomen. She has had recurrent attacks of pain in the lower abdomen for many years, and had appendectomy at Charity Hospital nine years ago for a so-called chronic appendix. The pain came back and she had it off and on ever since. For some time she has known that she had hypertension; in fact, since before the operation, and for six years she has noticed pigmentation of the skin which is not definitely related to exposure to the sun. There is no increased growth of the hair. She has had occasional precordial pain and cough, some shortness of breath, and has gained twenty pounds in the past five years. For a year or two she has noted some puffiness of the eyelids in the mornings and some swelling of the ankles in the evenings. She has had nocturia 1-2, but no hematuria. She has had headaches and epistaxis with menses, and has been on a "protein free" diet for several years.

The family history is irrelevant. She had measles and pertussis in childhood and "kidney trouble" for many years, with hypertension known for over nine years. Tonsillectomy was done at 26, and appendectomy at the age of 33. She had "influenza" six years ago. Menses have always been irregular and are now increasingly scanty.

She entered Touro in 1933, being treated in the clinic there for four or five years. At the time her complaint was pain in the legs. Her blood pressure on entry was 188/116, weight 81 pounds. At various times her blood pressure varied from 154/90 to 190/120. Four Wassermann tests were negative. They examined her blood, which showed: Phosphorus 2.5; calcium 10.2 in November, 1933; 8.6 in April, 1934; 10.5 in October, 1934. They took x-rays of her legs and reported, in May, 1934, a thickening of the cortex of tibia with rarifying osteitis of anterior surface, a small osteophyte being seen in the upper third of the tibia, suggestive of a possible luetic condition—hence the many Wassermann tests, which were negative. In May, 1936, another x-ray was made of the right tibia which showed a small bony prominence upon the medial aspect approximately two inches below the medial condyle, presenting somewhat the appearance of a flat exostosis. A dextrose tolerance test was made in 1934, 100 gm. of dextrose, showing 143 mg. (1 hr.), 117 (2 hrs.) and 115 (3 hrs.). In February, 1937, a P.S.P. test showed 40 per cent. In July, 1937, a Fishberg test showed 1010, 1012 and 1015. She discontinued going to the Touro Clinic in October, 1938, and then went to a private doctor who referred her to our clinic.

On November 28 she was admitted to our Clinic, at which time her temperature was normal, pulse 72, blood pressure 205/120, weight 101 pounds. Physical examination showed a short, dark woman, with ruddy, easily-flushed complexion. One was struck with the brownish pigmentation of the neck, chest, areolae and extremities. The hair on the head seemed to be quite normal; there was some hypertrichosis between the breasts and a male distribution of the pubic hair. There were no purple striae around the trunk or limbs. The thyroid was not enlarged. Fundi are negative except for shiny, thickened arterioles. The nose showed swelling of the turbinates almost entirely occluding meatus on left side. Occasional moist rales were noted at the bases of the lungs; the heart was not demonstrably enlarged; there were no murmurs; second aortic sound was increased. The abdomen was rather fat but not obese or pendulous. There was no pitting edema of the ankles. The laboratory work has been limited on account of the short time that she has been in the clinic. Her urine was acid, 1006, trace of albumin—one might call it a heavy trace; there was no glucose and microscopically it was normal; Wassermann was negative. Blood: red blood cells 4,950,000, hemoglobin 80 per cent, white blood cells 6,250, polys 73 per cent, large monos 3 per cent, small monos 24 per cent, NPN 41 mgm. per cent, globulin 3.08, albumin 3.58, total proteins 3.58. We did the blood proteins because she said that she had been on a protein free diet.

The diagnosis at Touro was: Obscure adrenal condition accompanied by hypertension; chronic osteitis of both tibiae, cause unknown. The diagnosis by her physician was: Chronic nephritis and menopausal syndrone. We were struck by the association of pigmentation and hypertension and the possibility that there might be some connection; apparently, they thought along the same line at Touro. She obviously has a kidney condition, probably nephrosclerosis and hypertension, but why the pigmentation? She does not present a very graphic picture of Cushing's syndrone, with no striking distribution of fat around the trunk nor purple striae, but she has in common with the syndrome hypertension and the tendency to gain weight and the osteoporosis. She has no amenorrhea, glycosuria, nor other factors that he describes. Personally, I have had no experience in adrenal tumors. I think that this is a distinct possibility which should be looked into further in this case.

Subacute Bacterial Endocarditis (Dr. Grace A. Goldsmith): Subacute bacterial endocarditis always presents an interesting problem. Early in the course of the disease, diagnosis may be extremely difficult. The prognosis is always grave and therapeusis remains a challenge to the clinician.

The patient I wish to discuss came to the Clinic on October 10, of this year, complaining of fever of four months' duration, weakness and weight loss. About June 15, 1938, he began to notice progressive fatigability. He consulted a physician who found that he was running fever varying between 99 and 100° every afternoon. He was hospitalized and remained in bed about five weeks, treatment consisting of the administration of sulfanilamide, which failed to help his condition. He returned home and, while able to be up and

about, was too weak to do any work. He had lost approximately 30 pounds in weight from the time of onset of his illness until he appeared at the clinic. There were no symptoms referable to any specific organ or system of the body with but one exception. On three occasions he had some rather unusual skin manifestations, two of the lesions appearing on the arms and one on the foot. The lesions were red, elevated and very painful, measured about an inch in diameter, lasted only a few days, and disappeared completely without scarring. The only other symptom was dyspnea on exertion, which was not a new complaint. At eleven years of age, the patient had tonsillitis and fever of one month's duration, diagnosed rheumatic fever with cardiac involvement. Since then, it has been necessary for him to limit physical activity considerably. The family history was interesting in that the patient's father died of pericarditis at the age of 42. His mother and one brother are said to have heart disease but the type is unknown.

Physical examination at the time of admission to this Clinic showed a tall, thin young man 23 years of age who appeared somewhat undernourished. His temperature was 99°, the pulse rate was normal and the blood pressure was 112/70. The skin was slightly pale, but a very careful search failed to reveal petechiae or evidence of the lesions noted in the history. There were no hemorrhages in the retina. There was no lymphadenopathy. The lungs were normal. The heart seemed to be slightly enlarged and a harsh presystolic and systolic murmur were heard in the mitral area. The mitral first sound was loud and the pulmonic second sound accentuated. Examination of the abdomen failed to reveal any abnormalities. The spleen was not enlarged. The extremities were entirely normal. There was no clubbing of the fingers. In spite of the paucity of positive findings, a tentative diagnosis of subacute bacterial endocarditis was made in view of the evidence of rheumatic heart disease with mitral stenosis, fever of four months' duration, and loss of weight. Many other diagnostic possibilities warranted consideration. The condition might have been active rheumatic fever, undulant fever, or some other infection plus rheumatic heart disease. He might have had a disease of the blood or an anemia secondary to a malarial infection.

Laboratory findings showed no abnormality of the blood except a mild secondary anemia. White cells numbered eleven thousand, which is about what one might except if he had subacute bacterial endocarditis. No large endothelial leukocytes, which are occasionally found in this condition, were seen. An x-ray of the chest showed that the size of the heart was about at the upper limits of normal; fluoroscopic examination failed to reveal any enlargement of the left auricle. Urinalysis was normal. The Wassermann reaction and the agglutination test for undulant fever were both negative. A non-hemolytic streptococcus was found on blood culture. The electrocardiogram was very interesting, showing an elevation of the S-T segment with inversion of the T-wave in Leads II and III. These changes were consistent with an old posterior infarction of the heart, but were more likely due to a toxic or infectious process. Embolism of the coronary arteries is extremely rare and there were no symptoms of coronary occlusion.

The patient was hospitalized at Touro and, while there, developed a number of petechiae in the buccal mucous membrane, particularly on the hard palate. The temperature ranged from 99° to 101°. The urine was examined frequently but no red blood cells were ever found. Blood cultures were positive on several occasions for Streptococcus viridans. He received two blood transfusions with some subjective improvement after the first but none after the second. He was also given sulfanilamide but in small doses only as he exhibited intolerance to large amounts. It is unlikely that benefit would ensue from the use of this drug since it has not been shown either in vitro or in vivo to be efficacious against Streptococcus viridans. A new pyridine derivative of sulfanilamide gives promise of being active against this microorganism and may be worthy of trial. Although this patient has been shown to have a subacute bacterial endocarditis, he has had few embolic phenomena. Petechiae are believed by some to be due to toxic damage to the capillaries rather than to emboli. Moreover these lesions have appeared only in the mouth. The patient has not had pain in the region of the spleen nor has that organ been palpable. There has been no evidence of emboli to the kidney, no tender fingers and toes, no clubbing of the fingers. In spite of few physical findings, the disease has been progressing rapidly and unfavorably. The prognosis in subacute bacterial endocarditis is grave, the mortality, according to Libman, being about 97 per cent. In occasional instances spontaneous recovery occurs. A few individuals become bacterial free and die of the cardiac or renal damage resulting from the infection.

Hepatomegaly and Diarrhea (Dr. Louis K. Levy): This patient, a 46 year old married woman, came to the clinic with the complaint of diarrhea. About four months ago, after having been in apparently perfect health, there was the sudden onset of diarrhea, which consisted of the passage of 10 to 14 stools per day. There is no history of fever or chills nausea or vomiting. During the entire time there has never been any period of constipation and the severity of the diarrhea has remained unchanged since the onset. On several occasions tarry stools have been noted and on two occasions the stool contained streaks of gross blood. During her illness the patient's appetite has been poor and she has lost some weight, the exact amount not being known, but apparently the weight loss has been slight. Pain of any description has not been present.

Examination reveals a well developed, well nourished, white female. The temperature and blood pressure readings are normal. There is no abnormal pigmentation of the skin, and the buccal mucosa reveals no stomatitis or atrophic changes. The sclerae show no evidence of icterus. The chest is clear. The upper margin of liver dulness is not distorted (also proved by x-ray examination). The liver edge is palpable just beneath the costal margin. The edge is smooth, not firm to the degree of hardness, and not tender. There is no tenderness on tapping over the lower right chest wall. The spleen is not palpable and there is no abdominal tenderness. On vaginal examination a hard, nodular, fixed mass, which is about the size of a large pecan, is palpable in the posterior fornix. On rectal examination some diminution of the pliability of the rectal mucosa is noted 4 cm. from the anus on the anterior rectal wall at the point where the cul-de-sac mass can be palpated. The proctoscope can be introduced with ease and the area of abnormal rectal mucosa is seen to be ischemic, hypertrophied, and contains within its extent a shallow ulcer 1 cm. in diameter which does not bleed. Reflexes are normal. Laboratory findings reveal negative blood Wassermann, normal urine, a very slight secondary anemia, and a normal white count. Several stool examinations reveal no pus, parasites, gross blood or mucus, or tubercle bacilli. Gastric analysis reveals free HCl of 0° and total acidity of eight.

In presenting the discussion on this case, I wish to bring to your attention the possible etiologic agents of the syndrome of hepatomegaly and diarrhea occurring in individuals in good nutritional state. I regret that, due to unforeseen difficulties, this case has not been completely worked up and a definite diagnosis cannot as yet be made.

Amebic dysentery with complicating hepatic abscess must be considered, but the absence of parasites in the feces, the absence of liver or thoracic wall tenderness, and the lack of evidence of the usual toxemia are against this diagnosis. Bacillary dysentery when producing this syndrome is usually accompanied by multiple pyemic liver abscesses with resulting marked toxemia. Pernicious anemia has reached the stage where the neurologic changes take place by the time diarrhea makes its appearance, and also buccal mucosa changes are present as a rule at this stage. When this syndrome appears in tuberculosis we think of a miliary affair with its marked toxemia. In cirrhosis of the liver diarrhea may be an initial manifestation but it is usually associated with swelling of the abdomen and legs. Also in cirrhosis light jaundice is the rule; the spleen is usually enlarged, and, lacking in this case, commonly there is a history of alcoholism. My tentative diagnosis is malignancy of the left side of the colon, there being no findings present in this case against this diagnosis. However, before such, or any diagnosis can be made, a gastrointestinal series, barium enema, stool culture, and, by all means, a biopsy of the diseased area of rectal mucosa should be done. Finally, if all findings are negative, I feel that a laparotomy is definitely indicated due to the presence of the mass in the cul-de-sac.

Jaundice (Dr. Adolph Ogard): Relating the patient's history only as it seems relevant to the present illness, we have here a 29 year old colored housewife who for approximately three years has had vague intermittent epigastric discomfort, usually occurring shortly after meals, occasionally associated with nausea, and usually being relieved by taking a small amount of sodium bicarbonate which she states produces the emesis necessary for relief. For eight or ten years she has noted a marked intolerance to fatty foods and associates their ingestion with flatulence, belching, and sometimes with nausea and vomiting. She had a gastrointestinal series in July, 1935, which showed no abnormalities.

Since that time, she has been attending several clinics for a number of conditions which are not related to the present illness that began on November 18, 1938, with the onset of generalized itching of the skin, without any associated skin eruption. One or two days previously she developed a common cold which persists at this time. There was a slight degree of anorexia and malaise. Two days later she noticed the onset of nausea immediately after meals, shortly afterwards developing an epigastric pain which was at first dull in character, localized in the epigastrium, but which became stabbing and radiated to both sides of the upper abdomen, and on one occasion to the right shoulder. Epigastric symptoms did not persist with any degree of severity more than 24 hours. Four days following the onset of symptoms she noticed that her skin and sclerae were yellow, her urine dark in color, and three days later that her stools were lighter in color than usual. There has been no change in bowel movements as regards frequency. She had no fever during the present illness save on one occasion in the clinic on November 29 when it was 99.8°.

Complete physical examination on December 1 was entirely negative except for an icteric tint to the skin and sclerae and some papillary atrophy along the tongue margins. Abdominal examination showed no tenderness, guarding, or palpable viscera. Her subjective symptoms have not been severe at any time but mild malaise and anorexia have persisted to the present time. Stools are becoming darker in color; urine remains dark, and the icterus has receded. She was advised to rest in bed, take a high carbohydrate, low fat, low protein diet, and has been taking a teaspoon of sodium and phosphate b.i.d. as a saline purgative. There is no history of the use of drugs. She had lost eight pounds in weight during the past two years. The following laboratory findings were noted: Red blood cells 5,000,000, white blood cells 5,500, polychromatophilia 68 per cent, eosinophiles

2 per cent, small monos 30 per cent; icterus index on November 29, 50; December 1, 50; December 5, 35; December 7, 30; hemoglobin 78.8 per cent Newcomer, mean corpuscular volume 82, reticulocytes 0.2 per cent; on November 29, urine positive for bilirubin (foam test) and December 5 positive for urobilin but not for bilirubin (chemically); Wassermann negative. Stool examination shows no abnormalities. On November 22 she had a gallbladder visualization by the oral method, which failed to visualize the gallbladder.

The differential diagnosis or causes of jaundice may be considered under three main divisions: (1) Excessive red blood cell destruction giving rise to more bilirubin than even a normal liver can excrete, such as occurs in pernicious anemia, sickle-cell anemia, congenital hemolytic anemia, paroxysmal hemoglobinuria, mismatched transfusion and phenylhydrazine poisoning, such as may also occur in lobar pneumonia, hemolytic septicemias, malaria and blackwater fever. The history of this patient and the normal blood picture rule out such a possibility in this instance. (2) Occlusion of the bile ducts, small or large, giving rise to regurgitation of bilirubin into the lymphatics and blood stream (a) from pancreatic or biliary calculi. The mildness of symptoms, lack of history of previous colic, slow onset of jaundice after what may be interpreted as colic, age of the patient, and the absence of enlargement of the liver which often occurs with acute obstruction, tend to rule out the diagnosis in this case; (b) obstruction from parasitic plugging is pretty well ruled out by the negative stool examination; (c) obstruction from scarring as may occur in luetic hepatitis or chronic cholangitis—ruled out by transient nature of jaundice and negative history of lues with negative Kahn; (d) obstruction from neoplasm, primary or secondary in the liver or biliary tract or from the external pressure of a neoplastic growth; jaundice is transient and not progressive, with a relatively small amount of weight loss and no anemia; with inability to detect any abdominal tumor or to postulate any possible primary source. (3) A third cause of jaundice may be that from actual damage of the liver cells with inability of the cells to excrete bilirubin, with its resultant accumulation in the blood stream: (a) acute yellow atrophy; the symptoms are far too mild and there is no history of any possible etiology; (b) infectious or catarrhal jaundice: (1) due to spirochetal etiology—the mildness of symptoms rules this out; (2) epidemic infectious jaundice; there is no history of contact. A sporadic case of infectious jaundice of unknown etiology is the diganosis.

Favoring this diagnosis are: (a) Mild symptoms preceding and during transient jaundice; (b) age of patient; (c) low white count with normal distribution; (d) urobilinogenuria signifies impaired liver function; failure of gallbladder to visualize may also be due to this factor.

The future course of the disease will confirm or refute this conclusion.

TRANSACTIONS OF ORLEANS PARISH MEDICAL SOCIETY

CALENDAR

January 3. Board of Directors, Orleans Parish Medical Society, 8 p. m.
Eye, Ear, Nose and Throat Hospital Staff, 8 p. m.

January 4. Radio Talk, Orleans Parish Medical Society, 8:45 to 9 a. m., WDSU.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.
Hutchinson Memorial Clinic Staff, 8 p. m.
Mercy Hospital Staff, 8 p. m.

January 5. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

January 9. Orleans Parish Medical Society. Installation of Officers, 1939, 8 p. m.

January 10. Eye, Ear, Nose and Throat Club, 8 p. m.
New Orleans Hospital Council, I. C. R. R. Hospital, 8 p. m.

January 11. Radio Talk, Orleans Parish Medical Society, 8:45 to 9 a. m., WDSU.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.
Touro Infirmary Staff, 8 p. m.

January 16. Hotel Dieu Staff, 8 p. m.

January 17. Charity Hospital Medical Staff, 8 p. m.

January 18. Radio Talk, Orleans Parish Medical Society, 8:45 to 9 a. m., WDSU.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.
Charity Hospital Surgical Staff, 8 p. m.

January 19. Clinico-pathologic Conference, Touro Infirmary, 11:15 a. m. to 12:15 p. m.

January 20. I. C. R. R. Hospital Staff, 12 noon.
New Orleans Dispensary for Women and Children Staff, 8 p. m.

January 23. Orleans Parish Medical Society, 8 p. m.

January 24. Baptist Hospital Staff, 8 p. m.

January 25. Radio Talk, Orleans Parish Medical Society, 8:45 to 9 a. m., WDSU.
Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.

French Hospital Staff, 8 p. m.

January 26. Clinico-pathologic Conference, Charity Hospital and L. S. U. Medical Center, 2 p. m.

January 27. L. S. U. Faculty Club, 8 p. m.

During the month of December the society held one meeting, Monday, December 12. The second meeting was dispensed with because of Christmas holidays. The thirteenth annual Stanford E. Chaille Memorial Oration was delivered Monday, December 12, by Dr. Porter P. Vinson, Professor of Bronchoscopy and Esophagoscopy, Medical College of Virginia, Richmond, whose subject was "Aids in the Early Diagnosis of Primary Bronchial Malignancy."

This meeting also served as the annual memorial meeting in respect to the members of the society who passed on during the year. The following names were read out by the secretary:

[redacted]

IN MEMORIAM

Dr. Charles A. Wallbillich	February 10, 1938
Dr. Earl A. Hogan	March 16, 1938
Dr. Caroline Mims	May 8, 1938
Dr. Frederick L. Fenno	July 20, 1938
Dr. Robert H. Potts	August 22, 1938
Dr. Albert S. Yenni	October 1, 1938
Dr. Louis A. Meraux	October 6, 1938
Dr. Lionel L. Cazenavette	October 15, 1938
Dr. J. Raymond Hume	October 29, 1938
Dr. William H. Seemann	November 19, 1938

Since the meeting Dr. John Signorelli died, December 13, 1938.

[redacted]

The annual election of officers was held Saturday, December 10. The following doctors were elected to serve for 1939: President: Dr. Cassius L. Peacock; first vice-president: Dr. Edmund L. Leckert; second vice-president: Dr. Lucien A. Fortier; third vice-president: Dr. Andrew V. Friedrichs; secretary: Dr. Gilbert C. Anderson; treasurer: Dr. Edwin H. Lawson; librarian: Dr. Donovan C. Browne. Additional members of the Board of Directors are Drs. Henry B. Alsobrook, George H. Hauser and Shirley C. Lyons.

On Saturday, December 10, 1938, the annual dinner of the society was held at 7 p. m. at Arnaud's. It is with regret that we report a very poor attendance at this function.

NEW ITEMS

Dr. Lucien A. LeDoux was appointed Councilor from Louisiana for the Southern Medical Association for a term of five years. Dr. LeDoux succeeds Dr. Arthur A. Herold whose term of office expired.

The following members have been named on various sections of the Southern Medical Association: Dr. Elizabeth Bass, Chairman of Section on Pathology; Dr. Merrill Beck, Secretary of the Section on Anesthesia; Dr. Ernest Carroll Faust was re-elected Secretary of the American Academy of Tropical Medicine; Dr. J. W. Reddoch, Secretary of the Section on Obstetrics; and Dr. Theodore Watters, Secretary of the Section on Neurology and Psychiatry.

Members appointed to serve on the board of the Guidance Center of the New Orleans Institute of Mental Hygiene are Dr. Rigney D'Aunoy, Louisiana State University Medical School, Dr. Frank R. Gomila, by Mayor Maestri, and Dr. John H. Musser, Tulane University School of Medicine.

Dr. John T. O'Ferrall was appointed a member of the Orthopedic Council of the Committee for the Celebration of the President's Birthday for the National Foundation for Infantile Paralysis.

Dr. George Hauser has been appointed as city bacteriologist in the position made vacant by the death of Dr. William H. Seemann.

Dr. George F. Roeling was appointed Superintendent of the City Hospital for Mental Diseases to fill the vacancy caused by the death of Dr. L. L. Cazenavette.

Dr. Ansel M. Caine has been elected a member of the Board of Directors of the American Society of Anesthetists for three years and has been elected an associate member of the Examining Board of the Fellowship Committee of the American Society of Anesthetists for six years.

Drs. Isidore Cohn, I. Mims Gage, Ambrose H. Storck, Joseph A. Danna, Alton Ochsner, James D. Rives, Curtis H. Tyrone and Roy W. Wright attended the meeting of the Southern Surgical Association at White Sulphur Springs, West Virginia, December 6-8.

Dr. John H. Musser attended the meeting of the American Council on Medical Education and Hospitals of the A. M. A., December 4, in Chicago.

The Graduate School of Medicine of the Louisiana State University met December 3-4 in Lafayette in the new Charity Hospital. This is the first program of a series to be given throughout the state. Dr. James T. Nix, Dean of the Graduate School, and Dr. P. H. Griffeth, Director of the General Extension Division of the University, are cooperating in this activity. On December 2-3, the following members presented papers: Drs. Charles Bloom, Charles Bahn, John F. Dicks, Rigney

D'Aunoy, Val H. Fuchs, James K. Howles, Daniel N. Silverman, Thomas B. Sellers and Henry W. E. Walther.

Dr. William Harvey Perkins succeeds the late Dr. William H. Seemann as President of the Tuberculosis and Public Health Association of Louisiana.

The following doctors were elected to membership during December: Active: Drs. Wallace C. Beil, Attilio V. Filizola, Frank R. Gomila, Jr., Richard T. Stephenson, and Carl N. Wahl. Interns: Drs. Alice E. Baker, Robert N. Caylor, Homer J. Dupuy, Jr., Frances Evans, Anthony Failla, John M. Lyons, Jacques A. Magne, Edward W. Nelson, Roland F. Phillips, Joseph A. Sabatier, Jr., Maurice E. St. Martin, John C. Suares, Joe D. Talbot, Bertha N. Wexler, John A. White, Jr., and Lawrence McC. Williamson.

Dr. Lucien A. Fortier attended the meeting of the Radiological Society of North America in Pittsburgh, November 27-December 3.

Dr. Leon J. Menville attended the meeting of the medical staff of the Baptist Hospital at Birmingham, December 13-14.

Drs. Albert E. Casey, Manuel Gardberg and Samuel B. Nadler read papers before the Southern Section of the Society for Experimental Biology and Medicine, December 16.

Drs. Neal Owens and Thomas B. Sellers were guest speakers before the Tri-County Medical Society, December 13, at Hazelhurst.

Drs. Edward L. King, Maxwell E. Lapham and Alton Ochsner addressed the Issaquena-Sharkey-Warren Counties Medical Society in Vicksburg, December 13. Dr. Lapham also addressed the third annual meeting of the Mississippi Public Health Association at Jackson, December 8-9.

Drs. Eugene B. Vickery and William A. Reed attended the meeting of the Southeastern Branch of the American Urological Association at Louisville, December 3.

The following officers of the New Orleans Urological Society were elected: Dr. Monroe Wolf, chairman; Dr. Hugh T. Beacham, vice-chairman, and Dr. James S. Davidson, secretary-treasurer.

Drs. Edwin H. Lawson, Shirley C. Lyons and Julius L. Wilson attended the meeting of the Southeastern Mississippi Medical Society in Hattiesburg, December 8-9.

Dr. Warren H. Hebert addressed the Proctology Society of the School of Medicine, University of Pennsylvania, Philadelphia.

TREASURER'S REPORT

Actual book balance 10/31/38	$3,470.27
December credits	774.56
Total credits	$4,244.83
December expenditures	608.55
Actual book balance 11/30/38	$3,636.28

LIBRARIAN'S REPORT

During November, 76 volumes have been added to the Library. Of these 29 were received by gift, 32 by binding and 15 from the New Orleans Medical and Surgical Journal. Titles of recent date are listed below.

The Library has loaned to doctors during November, 1145 books and journals, or more than two to each member of the Society. In addition, 888 books have been loaned to students for overnight use, making a total of 2,033 for the month. These figures do not include the great use of books and journals within the Reading Rooms.

Members of the staff have collected material on the following subjects at the request of physicians:

Arteriography
Socialized medicine
Herpetic keratitis
Bilateral foreign bodies in the tracheal tree
Treatment of pellagra with nicotinic acid
Gastroscopy
State medicine
Theophyllin in bronchial asthma
Relationship between diseases of the mouth and diseases of the chest
Dentritic keratitis

An exhibit on the History of Blood Transfusion has been prepared and has attracted many visitors to the History Room of the Library.

NEW BOOKS

Assn. for Research in Nervous and Mental Diseases: Acute Epidemic Encephalitis, 1931.

Assn. for Research in Nervous and Mental Diseases: Schizophrenia, 1931.

Abt, I. A., ed.: Yearbook of Pediatrics, 1930-1931.

American College of Physicians: Directory 1933, 1935, 1937.

Colorado State Board of Health: Cancer, 1938.

Harvey Society: Harvey Lectures, 1938.

Karsner, H. T.: Human Pathology, 1938.

Blacklock, D. B.: Guide to Human Parasitology, 1938.

Bentley, A. O.: Textbook of Pharmaceutics, 1937.

Burke, R. M.: Historical Chronology of Tuberculosis, 1938.

Henderson, Yandell: Adventures in Respiration, 1938.

Castellani, Aldo: Climate and Acclimatization, 1938.

Schlanser, A. E.: Practical Otology, Rhinology and Laryngology, 1938.

Henle, Jacob: On Miasmata and Contagia, 1938.

Crothers, Bronson: Pediatrician in Search of Mental Hygiene, 1937.

Mead, K. C. H.: History of Women in Medicine, 1938.

Behan, R. J.: Cancer, 1938.

Hegner, Robert: Big Fleas Have Little Fleas, 1938.

Walton, R. P.: Marihuana, 1938.

Betterman, Amos: Diary for Years 1868, 1873, 1893, 1909-10, 1938.

Gilbert C. Anderson, M. D.,
Secretary.

LOUISIANA STATE MEDICAL SOCIETY NEWS

CALENDAR

Louisiana State Medical Society	April 24-26, 1939	Alexandria.
New Orleans Graduate Medical Assembly	February 6-9, 1939	New Orleans.

PARISH AND DISTRICT MEDICAL SOCIETY MEETINGS

District	Date	Place
Second District	Third Thursday of every month	
Parish	**Date**	**Place**
East Baton Rouge	Second Wednesday of every month	Baton Rouge.
East and West Feliciana	First Wednesday in February	Jackson.
Orleans	Second and fourth Monday of every month	New Orleans.
Pointe Coupee	First Tuesday in March	
Sabine	First Wednesday of every month	

NOTICE

On account of financial difficulties, the firm that was printing the New Orleans Medical and Surgical Journal was obliged to go into bankruptcy. This untoward event will disturb somewhat the coming out of the Journal this month. It has been necessary to make a sudden change because no information had been given us concerning the financial troubles that the firm was having. Consequently, the material that was set up had to be entirely reset and, because of the unfamiliarity of the new printers with the job, bringing out the Journal on the first day of the month very necessarily has been delayed.

In addition to this dilemma of delay, it has been necessary to change to a certain extent the font and format. These changes have been restricted to a minimum so as not to cause a break in the typography of the present volume.

In the hurry of getting out the December issue of the Journal, in order to get it out of the printer's hands before they were taken over by the courts, a few copies have been misbound. If any of the readers find that their number of the Journal has this fault, we will be glad to exchange it for one that has been properly bound.

SECOND DISTRICT MEDICAL SOCIETY

The members of the Second District Medical Society were guests of a number of New Orleans physicians at a banquet held at the St. Charles Hotel on December 8. Dr. C. L. Peacock was the toastmaster and accredited himself splendidly. Dr. J. E. Clayton, of Norco, is president, and Dr. Fred S. Herrin, of Destrehan, is secretary-treasurer of the society.

The society had its beginning with four members, who met in Hahnville a few years ago, when Dr. D. N. Silverman, the Councilor for the Second District, Dr. P. T. Talbot, secretary-treasurer of the Louisiana State Medical Society, and Dr. Leon J. Menville, past-president of the Louisiana State Medical Society, founded the organization. It has now grown to a society of over thirty members who meet regularly once a month at the homes of its various members. Socialized medicine was discussed freely by several of the physicians, which resulted in a better understanding of this topic of momentous importance.

Fred S. Herrin, M. D., Sec.

BI-PARISH MEDICAL SOCIETY

The Bi-Parish Medical Society was called to order on December 7, 1938 by the president in the Rist Hotel. Visiting guests for the evening were Drs. P. T. Talbot and N. K. Edrington of New Orleans, and Mr. J. B. Rivers, representative of Mead Johnson and Company.

The election of officers for 1939 resulted as follows: Drs. T. H. Pargen, president; A. D. Long, vice-president; E. M. Toler, secretary-treasurer; E. M. Toler, delegate; E. M. Robards, alternate.

On motion, the following committee was appointed to draft resolutions relative to the death of Dr. J. B. Sewell, of Saint Francisville: Drs. E. M. Robards, C. S. Miller, and T. H. Pargen.

The scientific program was as follows: Medical pictures were demonstrated on the screen by Mr.

J. B. Rivers. Dr. P. T. Talbot read a most interesting and instructive paper on "Arthritis," which was discussed favorably by members present.

On motion a vote of thanks was extended to Dr. Talbot and Mr. Rivers for the excellent program that they presented to our society. A vote of thanks was given to Mr. and Mrs. August Rist for the preparation of the superb and delicious dinner.

On motion, Dr. S. S. Speer of Slaughter, Louisiana, was elected a member of our society.

The next meeting will be held at the East Louisiana Hospital, Jackson, the first Wednesday in February at 7:30 p. m.

E. M. Toler, M. D., Sec.

EAST BATON ROUGE PARISH MEDICAL SOCIETY

The regular monthly meeting of the East Baton Rouge Parish Medical Society was held Wednesday, December 14. The following officers for 1939 were elected: Drs. U. S. Hargrove, Baton Rouge, president; J. T. Lewis, Baton Rouge, vice-president; Cecil Lorio, Baton Rouge, secretary-treasurer; J. L. Beven, J. J. Noto, T. C. Paulsen, all of Baton Rouge, delegates.

Dr. I. S. Kahn of San Antonio, Texas, spoke on "Bronchial Asthma and Other Aspects of Allergy." This was a highly interesting and helpful paper, and was enthusiastically received.

U. S. Hargrove, M. D., Sec.

POINTE COUPEE PARISH MEDICAL SOCIETY

The regular meeting of the Pointe Coupee Parish Medical Society was held, December 8, at Morganza, with Dr. J. W. Plauche as host. The dentists of the parish were guests at the meeting and at supper.

A paper on "Infections of the Hand" was to have been presented by Dr. J. M. Mosely, but, in his absence, a round table discussion on the subject was instituted.

The following officers were elected for the year 1939: Drs. J. W. Plauche, Morganza, president; J. M. Mosely, Lakeland, vice-president; E. G. Durel, New Roads, secretary-treasurer; R. McG. Carruth, New Roads, delegate; F. F. Rougon, Oscar, alternate.

The next meeting will be held the first Tuesday in March.

E. G. Durel, M. D., Sec.

SABINE PARISH MEDICAL SOCIETY

A meeting of the Sabine Parish Medical Society was held December 8, 1938, at the Allen Sanitarium in Converse. The following officers were elected for the coming year: Drs. H. M. Prothro, Pleasant Hill, president; S. F. Fraser, Many, vice-president; O. L. Sanders, Converse, secretary-treasurer; L. H. Murdock, Converse, delegate; Dave Kaminsky, Fisher, alternate.

A program committee, consisting of Drs. R. B. Stille, Dave Kaminsky and O. L. Sanders, was appointed.

During the year 1939 meetings will be held the first Wednesday in each month.

O. L. Sanders, M. D., Sec.

RAPIDES PARISH MEDICAL SOCIETY

A regular meeting of the Rapides Parish Medical Society was held at the Baptist Hospital in Alexandria on December 5, 1938. The following officers were elected for 1939: Drs. J. T. Cappel, Alexandria, president; N. T. Simmonds, Alexandria, first vice-president; J. L. Woodall, Colfax, second vice-president; J. A. White, Jr., Alexandria, secretary-treasurer; J. A. White, Sr., H. O. Barker, M. H. Foster, all of Alexandria, delegates.

Dr. M. H. Foster presented a paper entitled "Solace for the Senescent." A film on lipiodol injection was also presented.

J. A. White, Jr., M. D., Sec.

RED RIVER PARISH MEDICAL SOCIETY OFFICERS, 1939

President: Dr. W. E. Wilkinson, Coushatta
Vice-President: Dr. W. B. Hunter, Coushatta
Secretary-Treasurer: Dr. W. W. Gahagan, Coushatta
Delegate: Dr. L. S. Huckabay, Harmon
Alternate: Dr. F. E. DePriest, Coushatta

TERREBONNE PARISH MEDICAL SOCIETY OFFICERS, 1939

President: Dr. S. F. Landry, Houma
Vice-President: Dr. M. F. Houston, Houma
Secretary-Treasurer: Dr. H. L. Haydel, Houma
Delegate: Dr. W. A. Ellender, Houma

WEBSTER PARISH MEDICAL SOCIETY OFFICERS, 1939

President: Dr. Wilkins McDade, Minden
Vice-President: Dr. S. M. Richardson, Minden
Secretary-Treasurer: Dr. B. A. Norman, Minden
Delegate: Dr. S. F. Martin, Minden

NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

From the unusual number of inquiries as well as enrollments which have been received at this early date, the impression is gained that the third annual New Orleans Graduate Medical Assembly, which will be held February 6-9, 1939, at the Roosevelt Hotel, will be the largest from the standpoint of attendance. It is also especially significant that a very considerable number of the inquiries have come from those who have attended previously. This has been especially gratifying to the sponsors of this intensive refresher course, because it indicates that the previous programs have supplied just what the busy practitioner has so long felt that he needed most.

The success of the previous assemblies has been an inspiration to introduce additional features, and this year eighteen physicians, each one an outstanding leader in his special field, will contribute one of the best planned courses yet attempted. The guest speakers who have been chosen are as follows: Dr. Harry J. Shields, Toronto, Anesthesia and Gas Therapy; Dr. N. C. Gilbert, Chicago, Cardiology; Dr. Clinton Welsh Lane, St. Louis, Dermatology; Dr. Chester M. Jones, Boston, Gastro-enterology; Dr. James C. Masson, Rochester, Gynecology; Dr. Paul H. Ringer, Asheville, and Dr. Cyrus C. Sturgis, Ann Arbor, Medicine; Dr. Franklin C. Ebaugh, Denver, Neuropsychiatry; Dr. M. Edward Davis, Chicago, Obstetrics; Dr. Harry S. Gradle, Chicago, Ophthalmology; Dr. A. Bruce Gill, Philadelphia, Orthopedics; Dr. William P. Wherry, Omaha, Otolaryngology; Dr. Wiley D. Forbus, Durham, Pathology; Dr. Frederick F. Tisdall, Toronto, Pediatrics; Dr. Arthur U. Desjardins, Rochester, Radiology; Dr. C. Gordon Heyd, New York City, and Dr. I. S. Ravdin, Philadelphia, Surgery; Dr. Robert Herbst, Chicago, Urology.

Each one of these nationally and internationally famous authorities will contribute liberally to the program and will be available for informal discussion of the problems of those who attend.

A small registration fee of $10.00, which covers all of the features, including four round table luncheons as well as the smoker, makes it possible to enjoy four full days of medical education, combined with recreation in the interesting City of New Orleans. Ample provision will be made for the entertainment of the ladies. A postal card addressed to the New Orleans Graduate Medical Assembly, 1430 Tulane Avenue, New Orleans, will bring interesting information concerning the program as well as other attractions.

AMERICAN COLLEGE OF SURGEONS

A sectional meeting of the American College of Surgeons will be held in Nashville, Tennessee, with headquarters at the Andrew Jackson Hotel, January 18-20, 1939. The following states will participate: Tennessee, Alabama, Arkansas, Georgia, Missouri, Florida, Mississippi, Louisiana, and Kentucky.

Dr. William D. Haggard is chairman and Dr. H. H. Shoulders is secretary of the Committee on Local Arrangements, which is making plans for an exceptionally interesting meeting.

Distinguished surgeons from all parts of the country who will address the sessions are Dr. Howard C. Naffziger, of San Francisco, President of the American College of Surgeons; Dr. George Crile, of Cleveland, Chairman of the Board of Regents; Dr. Irvin Abell, of Louisville, Vice-chairman of the Board of Regents and President of the American Medical Association; Dr. Alton Ochsner, Professor and Director, Department of Surgery, Tulane University, New Orleans; Dr. Frank E. Adair, New York City, Chairman of the Committee on Malignant Diseases, American College of Surgeons; Dr. Charles C. Higgins, Member of Staff, Cleveland Clinic; Dr. Frank K. Boland, Professor of Clinical Surgery, Emory University School of Medicine, Atlanta; Dr. James T. Nix, New Orleans, Professor of Surgery, Louisiana State University Medical Center; Dr. Fred W. Rankin, Surgeon, St. Joseph's and Good Samaritan hospitals, Lexington, Kentucky; and others.

Besides the operative and non-operative clinics in general surgery and the surgical specialties which will, as is customary at these sectional meetings, be held each of the three mornings in local hospitals, there will be on the afternoons of the first and second days a new feature—clinical assemblies. These will be held in the War Memorial. Four midday panel discussions will be held from 11:30 to 12:30 each of the three days. On Wednesday, January 18, the subjects will be: End-results of Gallbladder Surgery; Evaluation of Cancer Therapy; Thoracic Surgery, and Reduction in Appendicitis Mortality. On Thursday, January 19, the subjects will be: Hypertension and Surgery; Rehabilitation of Poor Surgical Risks; Abnormal Uterine Bleeding, and Urinary Obstruction and Infection. On the last day the subjects will be: Peptic Ulcer; Cancer; Toxemias of Pregnancy with Relation to Subsequent Health, and Interpretation of Surgical Lesions of the Colon and Rectum with Special Consideration of Their Ultimate Effect on Patients.

Hospital conferences and panel discussions on administrative and professional problems in hospitals will be held each day, and medical motion pictures covering general surgery and eye, ear, nose and throat surgery will also be shown daily. A Fracture Clinic is scheduled for Friday morning, January 20, and a Cancer Clinic for the afternoon of that day. The meeting will close with a session which will be open to the public on the subject of Conservation of Health.

Graduate training for surgery and the surgical specialties will be discussed at both the hospital and surgical sessions.

The medical profession at large, as well as hospital trustees, superintendents, nurses, and other hospital departmental personnel, will be interested in this meeting, at which there will be no registration charge. Members of the State Medical Association are most cordially invited to attend.

SPECIAL CONFERENCE

Dr. Lucien A. LeDoux represented the Journal Committee at the recent conference of presidents, presidents-elect, secretaries and editors of the state medical associations at the meeting of the Southern Medical Association in Oklahoma City on Tuesday, November 15. The purpose of this meeting was to acquaint informally the medical leaders of the different states with what the other was doing in regard to the many problems which confront the profession today, so that they could more or less adopt a common policy.

AMERICAN BOARD OF OPHTHALMOLOGY

The American Board of Ophthalmology announces an important change in its method of examination of candidates for the Board's certificate.

Examinations will be divided into two parts. Candidates whose applications are accepted will be required to pass a written examination which will be held simultaneously in various cities throughout the country, approximately 60 days prior to the date of the oral examination.

Examinations scheduled for 1939: Written: March 15 and August 5. Oral: St. Louis, May 15; Chicago, October 6.

Application forms and detailed information should be secured at once from Dr. John Green, secretary, 6830 Waterman Ave., St. Louis, Mo.

INTERNATIONAL COLLEGE OF SURGEONS

The officers of the International College of Surgeons are pleased to announce that the International College, in connection with the United States Chapter of that body, will hold its assembly in New York City at the Hotel Roosevelt, May 22-24, 1939.

Dr. Edward Frankel, Jr., of 217 East 17th St., New York City, has been appointed by the International officers as general chairman of this assembly. Any one interested in space for scientific exhibit will please communicate with him.

WORLD'S FAIR NEWS

For the first time in any international exposition, physicians and public health workers are to have at the New York World's Fair 1939 their own Professional Club, an incorporated organization, housed in a structure devoted to scientific and educational exhibits in their own field.

Membership in the Professional Club at present is open to accredited members of the American Medical Association and the American Public Health Association. It is expected that membership will be extended to include members of other professional groups.

MEDICAL COLLEGE OF VIRGINIA

The Medical College of Virginia, Richmond, celebrated Founders' Day marking the one hundred first anniversary of its founding on Thursday, December 1. Mr. Virginius Dabney, Editor of the Richmond Times-Dispatch, spoke on "Medicine in a Changing World." The exercises were preceded by academic procession of visitors, faculty, and members of the student body.

NATIONAL SOCIAL HYGIENE DAY

"Guard Against Syphilis" is the slogan of National Social Hygiene Day to be observed throughout America on February 1. The day will be marked by some 5,000 meetings to highlight present community campaigns against syphilis, according to Dr. Walter Clarke, executive director of the American Social Hygiene Association.

Evidences of interest in the forthcoming event, the third annual observance in the current attack on syphilis, point to a larger nation-wide demonstration than those of previous years, Dr. Clarke said. The association's offices in New York, as well as San Francisco, report increased activity and interest on the part of official and voluntary health and welfare agencies, civic groups, service clubs, women's organizations, churches, schools and upward of 1000 youth bodies.

A $15,000 gift to the National Anti-Syphilis Committee of the American Social Hygiene Association raises the total in its $500,000 appeal to $187,065 and will aid in promoting National Social Hygiene Day.

LAFOURCHE VALLEY MEDICAL SOCIETY

The Lafourche Valley Medical Society held its annual duck dinner at Raceland on December 21, 1938 at 7:00 p. m. The guest speakers were Drs. Leon J. Menville and J. K. Howles of New Orleans.

Dr. P. T. Talbot, Secretary-Treasurer of the State Medical Society, visited the fifth and eighth districts in the interest of the membership drive on December 2 and 3. He attended the meeting of the Fifth District Medical Society on December 2.

On December 7, Dr. Talbot was a guest at the Bi-Parish Medical Society meeting in Clinton, where he presented a paper on arthritis.

NEW MEMBERS

The following new members were admitted to the Louisiana State Medical Society during the month of December: Dr. P. P. LaBruyere, Jr., of Marrero; Dr. J. J. Massony, of Westwego, and Dr. J. A. Hendrick, Jr., of Shreveport.

Dr. Earl C. Smith, by request, addressed the Health Builders' Club on December 7, at their monthly meeting held at the New Orleans Hotel. The subject presented was "A Discussion of Appendicitis."

The International Postgraduate Medical Assembly of Southwest Texas, Inc. will be held in San Antonio on January 24-26, 1939.

The fourth annual Postgraduate Institute of the Philadelphia County Medical Society will be held in Philadelphia during the week beginning March 13, 1939. The subjects to be considered are those embraced by the terms blood dyscrasias and metabolic disorders.

The fifth annual meeting of the Mississippi Valley Medical Society will be held in Burlington, Iowa, September 27-29, 1939.

The Mississippi Valley Medical Society offers a cash prize of $100.00, a gold medal and a certificate of award for the best unpublished essay on a subject of interest and practical value to the general practitioner of medicine. Entrants must be members of the American Medical Association.

Dr. James B. Murphy, Chief of the Cancer Research Division of the Rockefeller Institute, New York, and Dr. Mont Rogers Reid, Director of Surgical Service of the Cincinnati General Hospital and Professor of Surgery at the University of Cincinnati, have been named as new members of the National Advisory Cancer Council for three-year terms.

The appointments were announced by Surgeon General Thomas Parran of the United States Public Health Service, who is ex-officio chairman of the Cancer Council. The new members of the council succeed Dr. Francis Carter Wood, Director of the Crocker Institute of Cancer Research, Columbia University, and Dr. James Ewing, Director of the Memorial Hospital, New York, both of whom were named as original members of the council on October 16, 1937.

AMERICAN BOARD OF OBSTETRICS AND GYNECOLOGY

The general oral, clinical and pathological examinations for all candidates, Part II Examinations (Groups A and B), will be conducted by the entire Board, meeting in St. Louis, Missouri, on May 15 and 16, 1939, immediately prior to the annual meeting of the American Medical Association. Notice of time and place of these examinations will be forwarded to all candidates well in advance of the examination dates.

Application for admission to Group A, May 1939, examinations must be on file in the secretary's office by March 15, 1939. Application blanks and booklets of information may be obtained from Dr. Paul Titus, Secretary, 1015 Highland Building, Pittsburgh (6), Pennsylvania.

HEALTH OF NEW ORLEANS

The Department of Commerce, Bureau of the Census, reports that for the week ending November 12, there were 142 deaths in the City of New Orleans, 98 occurring in the white and 44 in the negro race. This compares favorably with the week previous when 167 deaths occurred. Included in the report are 14 deaths in children under one year of age, divided practically equally between the two races. The next week, which came to a close on November 19, showed that there were 11 more deaths than the previous week, 4 less in the white and 15 more in the negro race. Infant deaths were only 10 this week, of whom three were white and seven negro. For the week ending November 26, 157 deaths were listed, 89 white and 68 negro. Infant deaths were 13 in number, divided seven white and six negro. For some inexplicable reason there was a very sharp increase in the number of people dying in New Orleans in the first part of December in the week which came to an end December 3. A greater number of deaths were listed for this week than has been noted in a long time, there being 124 white and 88 negro, for a total of 212. A part of this increase might be attributed to the increase in infant mortality which was 21.

INFECTIOUS DISEASES IN LOUISIANA

Dr. J. A. O'Hara, epidemiologist for the State of Louisiana, has furnished us with the weekly morbidity reports for the state, which contain the following summarized information: For the week ending November 19, the forty-sixth week of the year, as is customary syphilis led all reported disease, there being 176 cases recorded. Other diseases occurring in numbers greater than ten included 50 cases of measles, 36 of cancer, 28 each of pneumonia and malaria, 27 of pulmonary tuberculosis, 26 of scarlet fever, and 18 each of typhoid fever and diphtheria. The typhoid fever cases came for the most part from Caddo Parish, 4 cases, and Pointe Coupee, 5 cases. For the succeeding week, ending November 26, there were 109 cases of syphilis listed; 41 of measles, 38 of pulmonary tuberculosis, 28 of cancer, 24 of gonorrhea, 20 of diphtheria, 19 of scarlet fever, 16 of whooping cough and 12 of malaria. For the next week, which came to a close December 3, syphilis again was in the front with 112 cases. A mild epidemic of measles was present in the state, 83 cases being listed as contrasted with the five-year average of 5.6. Other diseases in double figures included 34 cases of pneumonia, 30 of diphtheria, 26 each of scarlet fever and whooping cough, 22 of pulmonary tuberculosis, 19 of cancer, and 11 of influenza. A case of typhus fever was reported from East Baton Rouge Parish and one of paratyphoid fever from Orleans. For the forty-ninth week of the year, there was a marked increase in the number of cases of syphilis, 242, and pneumonia, 65. Other diseases reported in double figures were 40 cases of gonorrhea, 26 of measles, 25 of pulmonary tuberculosis, 24 of cancer, 23 of scarlet fever, 13 of diphtheria and 12 of influenza. Of the rarer diseases, typhus fever was discovered in the Parish of Calcasieu and paratyphoid in Orleans.

JOHN SIGNORELLI
(1889-1938)

The innumerable friends and many patients of Doctor Signorelli were distressed to hear of his death on December 13, 1938. Doctor Signorelli had been sick for several weeks and a favorable outcome had been predicted, but a change for the worse resulted in his demise. New Orleans has

lost one of its outstanding men in the passing of this physician. Professor of Pediatrics in the Louisiana State University Medical Center and recently appointed to succeed the late Doctor Fenno as Medical Director of the Orleans Parish School Board, Doctor Signorelli had accomplished much in his forty-nine years of life. A keen diagnostician, a therapeutist of skill and a sympathetic physician, he had an enviable reputation in his specialty in New Orleans and in Louisiana. Doctor Signorelli was active in the Orleans Parish and Louisiana State medical societies. He held many positions, elective and appointive, in these organizations. He was a man of cheerful disposition, smiling, bright and happy all the time. The medical profession and his grateful patients have lost a true friend and a good doctor.

WOMAN'S AUXILIARY

Louisiana State Medical Society

President—Mrs. Frederick G. Ellis, Shreveport.
President-elect—Mrs. S. M. Blackshear, New Orleans.
First Vice-President—Mrs. C. A. Martin, Welsh.
Second Vice-President—Mrs. A. G. McHenry, Monroe.
Third Vice-President—Mrs. D. C. McBride, Alexandria.
Fourth Vice-President—Mrs. A. F. Hebert, New Orleans.
Treasurer—Mrs. O. C. Rigby, Shreveport.
Recording Secretary—Mrs. D. T. Milam, Monroe.
Parliamentarian—Mrs. A. A. Herold, Shreveport.
Corresponding Secretary—Mrs. C. B. Erickson, Shreveport.
Historian—Mrs. W. P. Gardiner, New Orleans.

Mrs. Frederick G. Ellis, president, called the meeting of the Executive Board in Shreveport, November 22. Matters concerning the year book, finances, spring convention, and public relations were considered. Preceding the executive meeting Mrs. Ellis served the guests luncheon, using a charming seasonal motif in the buffet and table appointments. Members of the Board attending were Mesdames D. T. Milam, C. P. Gray and A. G. McHenry, of Monroe; H. O. Barker, D. C. McBride, S. J. Phillips, R. B. Wallace and L. D. Gremillion, of Alexandria; A. A. Herold, C. E. Rew, J. E. Heard, C. B. Erickson, R. T. Lucas, C. R. Gowen, R. L. Simmons, J. L. Scales, and O. C. Rigby, of Shreveport.

CADDO PARISH

It is an annual custom of the Woman's Auxiliary to the Shreveport Medical Society to have special entertainment during the Christmas holiday season to which the husbands of the members are invited. This year the party was in the form of a delightful dinner which was given at the Shrine Club on Cross Lake. The dinner tables were cleverly arranged in the form of a three branched candelabra, each white covered branch ending in a square table covered with red cellophane, representing the flame of the Christmas candle. Down the length of the tables were groups of nine candle holders burning red tapers, wreathed with silvered sprays of ivy, and at each end were accent points of red poinsettias. Silvered pine tips with golden cones and silvered bells tied with red moiré ribbon were also effectively used in the table decorations.

The guests of honor were the advisors of the auxiliary, Drs. R. T. Lucas, R. L. Simmons and T. M. Oxford; also Mrs. F. G. Ellis, president of the Louisiana State Auxiliary and Mrs. C. E. Rew, president of the Caddo Parish Auxiliary, Mrs. J. T. Crebbin, past president, and Mrs. C. P. Rutledge, president-elect. Mrs. E. L. Gill was the charming toastmistress. A skit was cleverly put on by Mesdames Frank Walke, D. R. McIntyre, Douglas Kerlin, C. R. Erickson, W. B. Heidorn, T. E. Strain, R. L. Simmons and E. L. Gill. Gifts were distributed among the guests by a most jovial Santa Claus who was later identified as Dr. Dorf Bean.

Mrs. Robert Simmons,
Press and Publicity Chairman.

CALCASIEU PARISH

A luncheon meeting was held at the Majestic Hotel on Friday, November 18, 1938. Plans were discussed for preparation of Thanksgiving baskets for the needy families of Lake Charles, which were gathered and distributed by auxiliary members.

Interesting pictures were presented by Mrs. Frank Gibson who recently made a tour of Alaska and the northwest.

The December meeting was held at the Bel Community Hall on Friday, December 19, 1938. The ladies were delighted to have with them Mrs. F. G. Ellis, of Shreveport, and Mrs. J. D. Frazer of DeRidder. Mrs. Ellis, who is state president, gave a very interesting and inspiring talk on auxiliary work over the State of Louisiana.

The Calcasieu Medical Auxiliary won over the American Legion Auxiliary in a Radio Quizology Contest. Members of the auxiliary are still active in the program, meeting the different auxiliaries of Calcasieu Parish.

Mrs. L. L. DiGiglia,
Press and Publicity Chairman.

Respectfully submitted,
Mrs. Robert T. Lucas,
Press and Publicity Chairman.

BOOK REVIEWS

Dr. Colwell's Daily Log for Physicians, 1939: Colwell Publishing Company, Champaign, Illinois. Price $6.00.

This volume provides in a compact and concise manner all the bookkeeping forms that are essential for keeping an accurate account of professional transactions. The forms are simple, brief, and comprehensive. They are easy to understand and require no previous knowledge of bookkeeping.

A page for each day of the month allows space for the names of patients seen, the services rendered, and whether cash was received or a charge is to be made. At the end of each month, a summary gives at a glance the day by day record of cash and charge transactions. There are also other forms for summarizing office expenses, narcotics dispensed, surgical operations, social security taxes, personal receipts and disbursements.

Dr. Colwell's Daily Log needs no commendation to those who have already used it. It is a time saver for the busy doctor who is his own bookkeeper and a means of securing accurate financial records for the doctor who is dependent upon clerical assistants to do his bookkeeping.

PHILIP H. JONES, JR., M. D.

Clinical Medicine in Practical Medicine: By C. P. Stewart, M. Sc. (Dunelm.), Ph. D. (Edin.), and D. M. Dunlop, B. A. (Oxon.), M. D., F. R. C. P. E. Baltimore, William Wood and Co., 1937. Pp. 372. Price $4.00.

This little book deals with the performance and interpretation of a group of chemical tests which are widely used today in the diagnosis of disease entities.

The chapters on glycosuria, cerebrospinal fluid and albuminuria, and tests of renal function are very thorough and of decidedly instructive value. This book as a whole brings nothing new to the field. To the student and general practitioner the information obtainable in any standard textbook might be of more practical value than that obtained through reading this book.

ALBERTO PRIETO, M. D.

Disinfection and Sterilization: By Ernest C. McCulloch, M. A., D. V. M., Ph. D. Philadelphia, Lea & Febiger, 1936. Pp. 525. Price $5.50.

This appears to be the first comprehensive treatise dealing with the fundamentals of disinfection and sterilization. It contains important information on the germicidal values of disinfectants and their effects on many species of micro-organisms, with chapters on water purification, sewage treatment and pasteurization.

It should be an ideal reference book for bacteriologists, public health workers and clinical pathologists.

J. R. KRIZ, M. D.

Principles and Practice of Obstetrics: By Joseph B. DeLee, A. M., M. D. Philadelphia, W. B. Saunders Company, 1938. Pp. 1211. Price $12.00.

The new seventh edition of the great textbook which has been so popular with practitioner and student has been thoroughly revised. The illustrations are excellent; those in color are vivid and command attention. Those plates on version, however, are still in need of revision. They are misleading.

The chapter on anesthesia and analgesia is given the space it deserves. It calls attention to the fact that the pain and discomfort of childbirth today can safely be relieved to a greater extent than formerly. Sulfanilamide in puerperal sepsis is well discussed: "The use of this drug has spread over the world with more than the usual rapidity, because in many cases its effect has been truly dramatic, but as always over-enthusiasm has resulted in failures, and worse, many complications and not a few deaths are coming to light." And so throughout the book frank suggestions based on sound judgment and experience are freely debated.

Labor, normal and abnormal, its management, course and treatment passes before you with almost vivid animation as you read and study these illustrations. I am glad to see Professor de Snoo's experiment given a page as it illustrates rotation in the mechanism of labor in occipital presentation. This mechanism is so clearly described that were all general practitioners who do obstetrics to study this chapter, a sudden decrease in maternal and fetal mortality would result.

The editor and his associates are due the highest praise for their accomplishment.

GEORGE A. MAYER, M. D.

The Vitamins and Their Clinical Application: By W. Stepp, M. D., D. Kuhnau, M. D., and H. Schroeder, M. D. Translated by Herman A. H. Bouman, M. D. Milwaukee, The Vitamin Products Co., 1938. Pp. 173. Price $4.50.

This is a manual which presents tersely the important facts about the vitamins. There is a short sketch of the history followed by the chemical discussion of the vitamin in question and then the usual vitamins are included—A, B, C, D, as well as E and H. Succeeding the general discussion of the vitamins, which takes some 106 pages, there is a chapter on vitamins and human nutrition, another on the daily requirements for man, and lastly one on the antagonism of the vitamins. All the information is summarized in the appendix which devotes from one to two pages to the functions, possible results of deficiency and the results of absence of the vitamin in question. There is a well selected bibliography. Altogether the book may be recommended most highly, not so much to the vitaminologist, whose knowledge of vitamins

is extensive, but largely to the practicing physician who wants to know quickly more about vitamins than he knows at the present time.

J. H. MUSSER, M. D.

Aids to Histology: By Alexander Goodall, M. D., F. R. C. P. (Edin.). Baltimore, William Wood & Company, 1938. Pp. viii+151; figs. 26. Price $1.25.

This compend of histology, a volume in the "Students' Aid Series," was first published in 1911. Its treatment of the subject is cursory, in keeping with the usual compend style. There is a regrettable lack of evidence of the prevailing present-day emphasis on functional considerations, the material being almost entirely confined to bare description of histologic structure.

HAROLD CUMMINS, Ph. D.

Diseases of the Skin for Practitioners and Students: By George Clinton Andrews, A. B., M. D. Philadelphia, W. B. Saunders Co., 1938. Pp. 899. Price $10.00.

This text, an entirely revamped edition, is superior to its predecessor in so many instances that only a few can be designated in this short review. The chapters on roentgen rays, fungous diseases and tropical diseases are as complete as separate monographs. The addition of the latest known facts about vitamin deficiencies is concise. The chapters on hypertrophies and atrophies, lipoidoses, besides being instructive, are beautifully illustrated. The illustrations throughout this edition are selected with the greatest of care, portraying the usual, rather than the unusual, forms. The chapter on syphilis contains one hundred pages on the rational handling of the syphilitic. This is an excellent text for anyone interested in skin diseases whether student, general practitioner or specialist.

M. T. VAN STUDDIFORD, M. D.

The Biology of Arteriosclerosis: By M. C. Winternitz, M. D., R. M. Thomas, M. D., and P. M. LeCompte, M. D. Springfield, Illinois, Charles C. Thomas, 1938. Pp. 142. Price $4.00.

This small monograph is a beautifully illustrated study of the biology of arteriosclerosis. It is clear that the authors believe the lesions of arteriosclerosis are more than merely degenerative. The first part of the book consists of a study of the extent and functional significance of the vasa vasorum in the normal arteries. Their intricate and painstaking technic is described in detail. By the use of such methods, the authors found an extensive vascular network in normal blood vessels which they traced to three separate sources, namely the adventitia, the region of orifices of branches and directly from the lumen of the vessel. Furthermore, they found that the three origins for mural vessels and the same designs that result from their anastomoses not only are present but become greatly exaggerated in the diseased artery walls. They found that hemorrhage is but an exaggerated form of exudation and is common in the artery wall. It occurs in those locations which are the site of predilection of the arteriosclerotic lesions. In addition, they found that hemorrhages abound in those regions which are the site of origin of vasa in the normal arteries of man. From this, they predicate some relation between vascularity, hemorrhage and sclerosis.

There are, of course, a multiplicity of causes for hemorrhage which need not be mentioned here. While the authors do not consider hemorrhage and perhaps lesser exudations to be the only source of the materials that form atheroma, they are, nevertheless, potent contributing factors. The cause of arteriosclerosis is not to be found in a single factor. The pleomorphism of the process, its characteristic irregularity of distribution and rate of development are sufficient evidence of this.

In conclusion, the authors plead for an approach to this problem which, "based on recognition of the artery as a vascular or potentially vascular organ and therefore subject to the same pathologic processes to which other tissues are subject, may prove more fruitful than one in which lesions are regarded as primarily 'degenerative' or as the inevitable concomitants of age."

It would appear that any new approach to this subject is of the utmost importance even if it does nothing more than stimulate interest and research; therefore the reviewer recommends this volume to all those interested in arteriosclerosis.

RANDOLPH LYONS, M. D.

Pediatric Surgery: By Edward C. Brenner, M. D. Philadelphia, Lea and Febiger, 1938. Pp. 843. Price $10.00.

According to his preface, it is the author's purpose, in response to the request of his students, "to present a volume neither encyclopedic nor compendial in type, which may be of practical value to those engaged in the study and practice of pediatric surgery." He goes on to say that "illustrations have been freely employed, and bibliographies and theoretical discussions omitted." In spite of the author's denial of such intentions, the book leans toward the compendial type: A wide variety of surgical conditions is discussed in its fifty-two chapters, even though fractures, dislocations, and other orthopedic problems have been omitted.

In some centers pediatric surgery has been accepted as a surgical specialty, although in most localities it is still handled by the general surgeon. There is no special reason why it should not be. The surgical problems of children are much the same as the surgical problems of adults. What the general surgeon does need, however, is advice as to how surgical diseases are exhibited in children, and wherein their symptoms, signs, and details of management differ in children and in adults.

All of this material is in this book, but its significance is somewhat impaired because it is almost buried in the details of conditions not peculiar to childhood. In other words, if the book had been made more compact it would probably be more useful for the purposes we have suggested.

The subject matter is so presented that it is interesting and easily read. This, and the tendency toward compendial presentation already noted, should make the book popular with the undergraduate student. The practicing physician, seeking help in problems peculiar to children, will wish for the bibliographies which have been omitted because many of the subjects presented require further elucidation before the recommendations made can be put into practice.

The author's associates are responsible for special chapters on special subjects. There are seven chapters on pediatric urology by C. G. Bandler and A. H. Milbert, and eight on neurologic surgery by J. E. Scharff. The chapter on the thorax by L. R. Davidson contains a particularly good discussion of empyema, as well as an excellent summary of the indications for and the methods used in collapse therapy. H. S. Vaughan has written the chapter of cleft lip and palate, and the description of operative procedures is very well done. Other special chapters include one on anesthesia by T. D. Buchanan, and one on blood transfusion by L. J. Unger.

The remaining chapters are by the author, and those on the colon, rectum, and anus should be singled out for special mention. The chapter on hernia contains much information useful to the general surgeon. Many authorities will disagree with the author on the conservative treatment he advocates for acute hematogenous osteomyelitis, though others will support him.

This book can be recommended to students not only for the knowledge of pediatric surgery they may derive from it, but also for the knowledge of general surgery they must inevitably secure from a book containing so many essential facts. The general surgeon whose knowledge of pediatric surgery is limited will find it a useful book for ready reference, though the more experienced surgeon will find in it little he does not already know. The author is to be commended for the emphasis he has placed on the importance of preoperative and postoperative care, and for his effort throughout the book to instill in his readers the maxim he has selected for his introduction, "In corpore pueri non est locus chirurgiae violentae."

The 293 engravings are in the main excellent.

SAMUEL A. ROMANO, M. D.

Practical Otology, Rhinology and Laryngology: By Adam Edward Schlanser, M. D. Philadelphia, Lea & Febiger, 1938. Pp. 315. Price $4.50.

This is a rather condensed textbook of 315 pages with information which is of value primarily to the beginner in otolaryngology, although there is much that will be of interest to the advanced student. The clinical side of the subject is emphasized and historical data, anatomy, physiology and detailed symptomatology have purposely been largely omitted for the sake of brevity. As the book represents Colonel Schlanser's years of army experience, it will be of particular value to the army medical officer.

H. L. KEARNEY, M. D.

A Pediatrician in Search of Mental Hygiene: By Bronson Crothers, M. D. New York, The Commonwealth Fund, 1937. Pp. 271. Price $2.00.

This volume is intelligently prepared by a pediatrician attached to the Harvard Medical School, likewise the Children's and Infants' Hospital in Boston, aside from his vast experience and opportunities afforded him to be conversant with mental hygiene and child guidance problems.

There are three parts: Part One deals with mental hygiene in the practice of medicine; Part Two deals with mental hygiene in the teaching of medicine; Part Three discusses "Towards Meeting the Pediatrician's Responsibility."

The volume is a plea for the proper intellectual fundamental approach to mental hygiene in pediatrics; likewise, a rebuke to the specialist who would thin out his medicine to the extent of attaching other medical entities under the guise of his chosen specialty. Well does he state that the pediatrician should not be a neuropsychiatrist, but does plead, however, that the pediatrician should understand full well the mechanisms and motivations which underly the unusual and bizarre reactions found in children who may become potential problem children or subjects for child guidance care.

This book should be collateral reading for the general practitioner, neuropsychiatrist, social worker, and the lay trained individual who is attempting to enter agencies dealing with an institute of human relations and children's problems.

WALTER J. OTIS, M. D.

Diseases of the Nose, Throat and Ear, Medical and Surgical: By William Lincoln Ballenger, M. D., F. A. C. S. 7th ed. rev. by Howard Charles Ballenger, M. D., F. A. C. S. Philadelphia, Lea and Febiger, 1938. Pp. 1030. Price $11.00.

It takes one back a good many years to remember the first edition of "Diseases of the Nose, Throat and Ear" by William Lincoln Ballenger in 1908. The continued popularity of this reference work is a deserved tribute to that illustrious otolaryngologist. Ten years after his death, December 21, 1915, the next (fifth) edition was published under the direction of Howard Charles Ballenger. The present (seventh) edition is the third revision by Howard Charles Ballenger.

While, in general, the form of the older work has been maintained, new chapters have been added

on physical therapy in otolaryngology, deep neck infections and petrositis.

A separate chapter has been devoted to benign neoplasms of the nose, throat and larynx. Malignant neoplasms of the larynx, throat and nose have been combined into one chapter. In a strictly clinical reference work such as this, the reviewer prefers the older arrangement. The section on bronchoscopy has been ably revised by Gabriel Tucker and Chevalier L. Jackson.

One hesitates to criticize comparatively unimportant details in an otherwise splendid book, but typographical errors are annoying. Figure 289 is captioned "paralysis of the arytenoids," when paralysis of the arytenoideus is meant. Petrositis is so spelled in the preface and thereafter is spelled petrousitis. The syndrome of Tapia is called the syndrome of Topia.

H. L. KEARNEY, M. D.

A Textbook of Pharmaceutics: By Arthur Owen Bentley. Baltimore, Wm. Wood & Co., 1937. Pp. 1001. Price $5.00.

This volume is by its very nature not only a textbook but also a handbook. Because it is the collaborative effort of several British authors it shows the British approach to the problem of teaching pharmaceutics.

The inclusion of ample discussion of pharmaceutical history and chemical engineering practice in pharmaceutical manufacture enlarges the usefulness of the book. The sections containing these discussions confirm one's understanding of the essential dignity of the pharamaceutic art and sharpen his appreciation of the problems of large scale manufacture of drugs. The cuts and line drawings of chemical engineering tools are unusually clear. They portray instruments which are actually in use at the present time.

The authors display no inclination to relegate to a place of unimportance in the book difficult concepts which have arisen in the sciences upon which pharmaceutics is based. This represents, however, merely another phase of their implicit intention to make the book complete and dignified.

There is, of course, the usual routine treatment of standard topics in pharmaceutics according to the British viewpoint. Contemplation of the British procedures serves to intensify an intellectual "hands across the sea" attitude.

The reviewer is impressed by the very extensive treatment of pharmaceutical abbreviation. He is happy to know where he can find the meaning of that obscure abbreviation the next time it rears its ugly head.

This book cannot fail to be useful to American pharmacists, pharmacologists, and pharmaceutical chemists.

JAMES C. RICE, M. D.

Materia Medica, Pharmacology and Therapeutics: By Walter Arthur Bastedo, Ph. M., M. D., Sc. D., F. A. C. P. Philadelphia, W. B. Saunders Company, 1937. Pp. 778. Price $6.50.

Drastic revision has brought this book into line with the most recent advances in pharmacology and the practice of medicine. Drugs such as acetyl choline, glycine, metrazol, sulfanilamide, vitamins, hormones, atabrine, cyclopropane, evipal, marihuana habit, toxic action of aminopyrine, toxic action of dinitrophenol for obesity, and carbon tetrachloride, are discussed fully. Practical pharmacy, and prescription writing are covered in essential outline.

The book is of particular value to practicing physicians, but will continue to be useful also for medical students.

CLYDE BROOKS, M. D.

Adventures in Respiration: By Yandell Henderson. Baltimore, The Williams and Wilkins Co., 1938. Pp. 316. Price $3.00.

Here in the compass of a small book is a brief story of the life work of a great scientist as told by himself. To Yandell Henderson and to the late John Scott Haldane of Oxford, to whose memory this work is dedicated, we owe, more than to any others, our knowledge of the respiratory apparatus. The facts are presented, with little theory or system, as a simple story of adventures, all pleasant in retrospect, but some filled with the anxious moments of an explorer in strange and uncharted regions.

The main theme of "The Adventures" is the problem of asphyxia. Shock, the failure of the circulation, the fallacy of asphyxial acidosis, the control of breathing, mountain sickness, carbon monoxide asphyxia, how breathing begins at birth, asphyxia and resuscitation of the newborn, postoperative depression, and the functional factor in pneumonia are the chief sub-themes used to show how important carbon dioxide is to well regulated and efficient respiration.

The style is delightful, clear, logical and forceful, characteristics all too uncommon in the writings of scientific men. The book is warmly recommended to clinicians, young and old.

HENRY LAURENS, Ph. D.

The Harvey Lectures, 1937-1938: Baltimore, The Williams and Wilkins Co., 1938. Pp. 275. Price $4.00.

This volume contains the lectures given during the last year under the auspices of the Harvey Society of New York and under the patronage of the New York Academy of Medicine. As the publication announcement sets forth "edited and illustrated for publication, the book provides a record of some of the most recent important research work by eight distinguished authorities."

The lectures include splendid, brief resumés of experiments on the nature of the visual process by Selig Hecht, the function of the lymphatic system by Cecil K. Drinker and the cortical representation

of somatic sensibility by Philip Bard. Einar Lundsgaard presents data which throw doubt on the validity of the assumption that the aerobic metabolism of active muscles is a pure carbohydrate metabolism. The lecture by F. C. Koch on the chemistry and biology of male sex hormones is profusely illustrated with graphs and tables showing the interrelationships of the androgens with each other and with other hormones. The discussion of water metabolism by John P. Peters is an unusually lucid presentation of a difficult subject. Particularly timely and stimulating is the description by Wendell M. Stanley of his important and far-reaching experiments leading to the isolation of the tobacco mosaic virus and his discussion of virus proteins, and the account by Harry Goldblatt of his work on the induction of experimental hypertension by renal ischemia.

This small volume is highly recommended to those interested in keeping abreast of the developments occurring so rapidly on the various fronts of medicine.

H. S. MAYERSON, Ph. D.

A History of Women in Medicine: By Kate Campbell Hurd-Mead, M. D. Haddam, Conn., The Haddam Press, 1938. Pp. 569. Price $6.00.

There is a legend in Israel that when the Lord contemplated the creation of Eve, after much deliberation, He decided to fashion her from Adam's rib in order that she might be modest and content, figuratively as well as literally, to conceal herself from public notice so that attention might not be diverted from her mate. And man wallowing in his egoism concluded that the woman was inferior mentally and physically. He did not know then, it is difficult for him to remember now, that the physical endurance and the mental capacity of woman is on a par with that of man. For years we have read histories of medicine and noted the notable achievements of men in the great unfolding of the drama of medicine from the dawn of history to the history of today's dawn. It has seldom occurred to us to ask the question,—what about the contribution of women to medicine? Granted that we have accepted the fact and are cognizant of the innumerable accomplishments and the tremendous achievements of the modern woman in medicine and the allied sciences, we remain dubious of what the woman of the past did to further the healing art. The fault, in great part, was due to the fact that history was written by men about men for men. Now, a new phenomenon has appeared on the horizon of medical history, a book by a woman and a physician about women and physicians for men and women, lay and professional. The viewpoint is novel, diverting and instructive. Here women stalk the stage and man is most often a mere prop. Truly one is stirred at the remarkable recital of Dr. Mead's "History of Women in Medicine." One can only wonder at the superiority of those women who had to fight superstition, bigotry and jealousy to gain and maintain their position in the face of what many men would have considered insurmountable obstacles. But the myth of the weaker sex is again exploded as the author repeats ad infinitum how women attained the heights. That this book will endure none can doubt. It is already a classic and it more than fulfills the requisites of the poet who said:

"Books should to one of these four ends conduce
For wisdom, piety, delight, or use."

In wisdom it abounds. Many years of effort went into its making. Travel and correspondence, world-wide in scope, helped to fashion it. Extensive reading and years of practice served to give it those elements that constitute the true landmarks of progress in medical literature. Every page reflects its piety. Imbued with the high purpose of the medical calling and feeling it her holy duty to proclaim the noble deeds of women in the fight against disease and death through the centuries, Dr. Mead writes in a strain that inadvertently labels the book as a sincere and true document and thus truly spiritual in nature. The delight to be derived from the perusal of this volume, is a matter of moment. Here is a book replete with medical lore to which all doctors must thrill. The status of medicines and drugs from primitive times to the close of the eighteenth century with the concomitant story of the social and economic, as well as the political and medical life cannot fail to afford sheer pleasure to the reader. Many curious facts from sources long forgotten are recalled in order that one may the better appraise the real value of the skill of medical women; the familiar male landmarks abound so that the reader may not lose himself in the hitherto little known or explored terrain of the feminine physician. Most interesting illustrations are interspersed among the pages.

Finally, the use of the book,—its value as a book of reference. Here one can only wonder at the persistence of the erudite scholar who for forty years labored to bring forth a book that at once establishes it as unique in the field of medical history even as it is the first pioneer effort in a most important subject. The facts are well authenticated and numerous documents and sources, so dear to the heart of the student, are an important part of the volume. It has been said that "there is no book so bad but something good may be found in it." I should like to paraphrase this by stating that there is no book so good but something bad may be found in it, and, briefly, I would remark that some authorities may find blemishes. If they exist I recognize them not and in my blissful ignorance I am happy not to be able to find the navel that bespeaks human imperfection. The author states that a second volume, which will bring her story to the present day, is forthcoming. It is bound to prove a welcome companion to a most worthy book. Truly one can say of Dr. Mead and her book that here are two great factors happily

met: The labor is worthy of the laborer even as the laborer is worthy of the labor.

I. L. ROBBINS, M. D.

Climate and Acclimatization: By Sir Aldo Castellani, K. C. M. G., D. S. C., M. D., F. R. C. P., Count of Kisymaio. London, John Bale, Sons & Curnow, Ltd., 1938. Pp. 198. Price 10/.

This is an all-inclusive, pocket-sized book of concise lore on the effects of climate on man, especially on the white man in the tropics.

The Count of Kisymaio is a man of profound learning and ripe experience in medicine, and particularly in tropical medicine. Therefore, when he was in charge of all military and civilian medical service for Italy during the Ethiopian war, he achieved an unprecedented record for good health and low mortality among the Italian troops and civilians.

Although the Count insists that his book is not an exhaustive work, yet it appears adequately to cover the essential phases of geography, meteorology, physiology, pathology, so far as these bear on the effects of climate on man. The book presents much knowledge derived from many of the more important documented works on the subject, a bibliography of which is included. The main body of the book, however, is derived directly from the Count's rich personal experience gathered in long sojourns in many tropical countries.

The student of climatology or tropical medicine, or anyone living in the tropics, or anyone who expects to go there, will find the book extremely interesting and profitable.

CLYDE BROOKS, M. D.

Hernia: By Leigh F. Watson, M. D. St. Louis, C. V. Mosby Co., 1938. Pp. 591. Price $7.50.

This book is a fine evidence of the value of text on certain relatively restricted fields. It is true that one seeking information on hernia might obtain all the information contained in this book from numerous sources, but certainly, if one is seeking concentrated information on hernias of any type, here is an excellent place to find it. The author not only considers the different types of hernias, such as inguinal, umbilical, femoral, obturator, sciatic, diaphragmatic, internal hernias, but he describes in detail the different methods of treatment. At the beginning of each chapter is an excellent historical resumé of the subject. Herein are found such interesting facts as that Bassini is not the author and the originator of the so-called Bassini hernioplasty. The author says that Marcy described the epoch making technic in a paper he read before the International Medical Congress, London, in 1881.

Perhaps the most significant part of this book, a distinct addition to the first edition which appeared in 1924, is the discussion of the injection treatment of hernias. It is surprising that the author, who has had considerable experience, endorses without equivocation the injection treatment of properly selected hernias. The injection treatment is suitable for 90 per cent of the patients. Comparative figures showing recurrence after operative methods and after injection treatment make the latter seem equally as good if selected cases only are injected. By injection treatment, small indirect inguinal hernias gave 94 per cent cures and large indirect inguinal hernias gave 85 per cent. Ample space and sufficient excellent drawings are given to indicate technic of all methods.

The style has a friendly and intimate simplicity making the text easy to read. This is a splendid book.

HOWARD R. MAHORNER, M. D.

PUBLICATIONS RECEIVED

Harvest House, New York City: Feminine Hygiene in Marriage by A. F. Niemoeller, A. B., M. A., B. S.

Light and Life Press, Winona Lake, Indiana: Doctors, I Salute! by Emilie Conklin.

Shaw Publishing Company, Washington: Drug Addicts Are Human Beings by Henry Smith Williams, M. D., B. Sc., LL. D.

Charles C. Thomas, Springfield, Illinois: Classic Descriptions of Disease by Ralph H. Major, M. D.

William Wood and Company, Baltimore: Physical Diagnosis by Richard C. Cabot, M. D., and F. Dennette Adams, M. D.